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DEDICATED TO

OUR FRIEND

GEORGE FETTEROLF, A. B., M. D., Sc. D.

ASSISTANT PROFESSOR OF ANATOMY IN THE UNIVERSITY OF PENNSYLVANIA
TO WHOM WE ARE INDEBTED FOR MOST OF OUR ANATOMIC SECTIONS
WITHOUT WHOSE CO-OPERATION, CORDIAL AND SELF-EFFACING,
OUR BOOK WOULD LACK WHAT IS PROBABLY ITS
MOST CHARACTERISTIC FEATURE.

PREFACE TO THE SECOND EDITION

THE exhaustion of the first edition of this book, in less than two years, may be taken as evidence that it supplied a need. The present edition contains descriptions of several conditions previously omitted. Among these may be mentioned—Spirochetal Bronchitis, Influenza, Streptococcus Empyema, Chronic Inflammatory Conditions of the Lungs of Uncertain Etiology, Calcification of the Lungs, Pneumopericardium, etc. In addition some of the original material has been recast, in part or completely so. In every instance the effort has been made to incorporate in the original text such additions to our knowledge as indicate a distinct advance.

The authors welcome this opportunity to express to old and new made friends their gratification over the cordial reception that was accorded the first edition; and also to venture the hope that the second edition will merit the same kindly greeting.

THE AUTHORS.

PHILADELPHIA, PENNA.
January, 1920

PREFACE

To write a practical book on the physical diagnosis of the heart and lungs in health and disease, has been the aim of the authors. We have attempted to omit everything not of practical diagnostic use, and to condense methods of secondary importance.

More than is the custom has been written on the subject of diagnostic acoustics, because we believe that only through the comprehension of the laws of sound production and transmission can the results of percussion and auscultation be intelligently interpreted.

We have endeavored to teach as much as possible by means of illustrations. Of these, many are photographs of frozen sections from the cadaver, previously hardened in formalin, so that the anatomic relations of the tissues remain as during life.[1] We have found these specimens invaluable in our own teaching; and the book has been written in the hope that the photographic reproductions of our sections may be useful to others.

We take pleasure in acknowledging our great indebtedness to Dr. George Fetterolf for the frozen sections; to Dr. J. Claxton Gittings for assistance in the preparation of the Section on Pediatrics; to Dr. Edward B. Krumbhaar for the Chapter dealing with the electrocardiogram; to Professor Richard Geigel,[2] and to Dr. Charles M. Montgomery,[3] from whose articles upon acoustics in diagnosis, much information has been gleaned. We are also indebted to Miss Eleanor A. Cantner for the production of some of our drawings. We are further under obligations to Professor William M. L. Coplin for permission to photograph numerous specimens in the Museum of Pathology of the Jefferson Medical College, as well as to Dr. David R. Bowen and to Dr. Henry K. Pancoast for our radiograms.

THE AUTHORS.

PHILADELPHIA, PENNA.

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[1] The photographs, both clinical and anatomical, were with negligible exceptions, made by Dr. George W. Norris. Many of the pictures illustrating diseases of the heart and aorta have been reproduced from "Studies in Cardiac Pathology" by George W. Norris.

[2] GEIGEL, RICHARD: "Leitfaeden der Diagnostischen Akustik." Stuttgart, 1908.

[3] MONTGOMERY and ECKHARDT: "Pulmonary Acoustic Phenomena." Tenth Annual Report of the Henry Phipps Institute, Phila., 1915.

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PART I

THE EXAMINATION OF THE LUNGS

BY GEORGE W. NORRIS, A. B., M. D.

CHAPTER I

PHYSICAL DIAGNOSIS

Physical diagnosis consists in employing our senses—sight, touch, and hearing—to determine the condition of the tissues. These are altered in character by many pathologic states. They may become more solid or less, may contain more or less of fluid or of air than normal, their elasticity may be increased or diminished. Again, it may be that organs become larger or smaller than normal, or are shifted more or less out of place. Such alterations we can often demonstrate by means of physical signs. The data thus obtained, used in conjunction with a knowledge of the patient's history, and symptoms, together with a familiarity with the pathology, often permit us to estimate very accurately the nature, character, location and extent of the disease from which the patient is suffering. "The significance of morbid signs relates immediately not to diseases, but to the physical conditions incident thereto. Signs are not directly diagnostic of particular diseases" (Flint). The methods employed in physical diagnosis are: *inspection, palpation, percussion*, and *auscultation*. These methods are frequently combined with mechanical, chemical, electrical, microscopic and bacteriologic examination, as well as with the data obtained by means of the X-rays.

INSPECTION

Although seemingly the most obvious, the simplest and the easiest of the four methods mentioned, accurate, useful and skilled inspection is in reality often the most difficult to acquire and the last in which the practitioner becomes proficient. It is in this method especially that the seasoned physician far excels his younger confrère. This is in part due to carelessness on the part of the younger man, but is perhaps even more due to the fact that the senior has become accustomed to make note of many items at a glance, and also that he has learned not only what to look for, but where to look for it, and how to read the facts which are presented before his eyes. He has acquired the faculty of seeing with the mind as well as with the eye. "We can only see what we have learned to see." Corrigan's remark is still as apt as the day it was uttered: "The trouble with most doctors isn't so much that they don't know enough, as it is that they don't *see* enough!" We feel

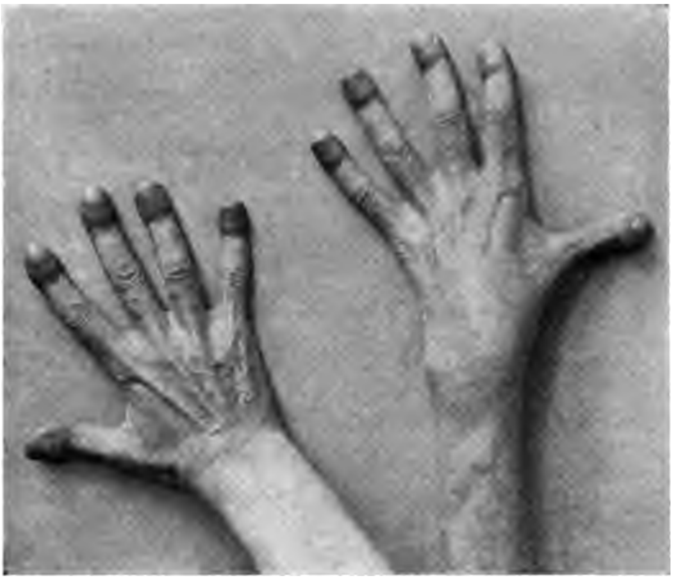


Fig. 1.—Clubbing of the fingers due to congenital heart disease.

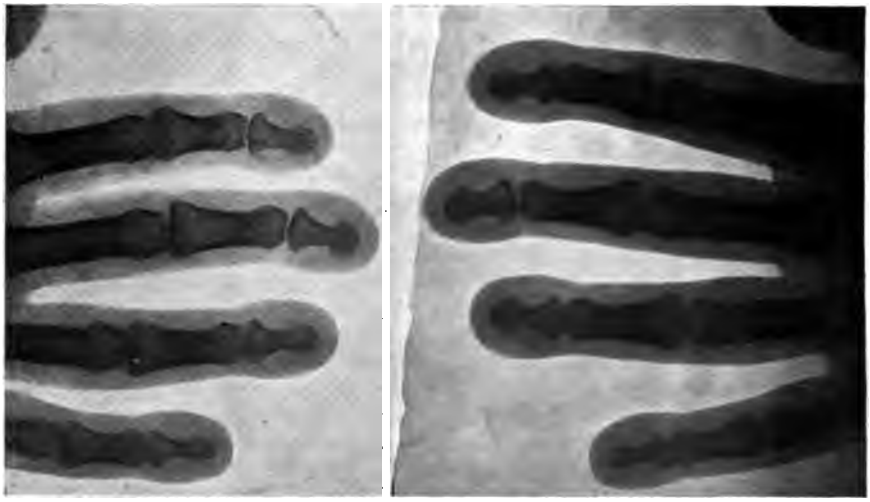


Fig. 2.—Radiogram of the fingers depicted in Fig. 1, showing not only hypertrophic changes in the soft tissues, but also new bone formation in the distal phalanges (pulmonary osteo-arthropathy). "Simple clubbing of the fingers and secondary hypertrophic osteo-arthropathy should be considered as identical, the former representing an early stage of the latter."

that we cannot over-emphasize the importance of careful, intelligent inspection.

The Examination of the Lungs.—For the purposes of examination, the body, especially the chest and abdomen, must be stripped. The light should be good. Its source, as to whether it falls directly, obliquely or vertically, upon the patient, must often be varied. Many physiologic and pathologic conditions can be seen only with oblique illumination. It is therefore desirable to have the patient first face the window, later turn his side toward it. In the latter position most of the shadows become intensified.

The following points are especially to be noted:

GENERAL INSPECTION

General appearance, posture, gait, facial expression, nutrition, color.

Absolute symmetry is unknown. As a general rule the right side of the body is better developed than the left. The right chest is about 1½ inches larger in circumference than the left. The spine curves toward the right, the right arm is longer and the corresponding shoulder is often narrower and lower. There are, of course, well-marked differences which depend upon: (*a*) *Sex:* These involve the bones, the pelvis, the genitalia, the panniculus adiposus, etc. (*b*) *Age:* In the *child*, the ribs are more horizontal, the heart and liver larger, the lungs

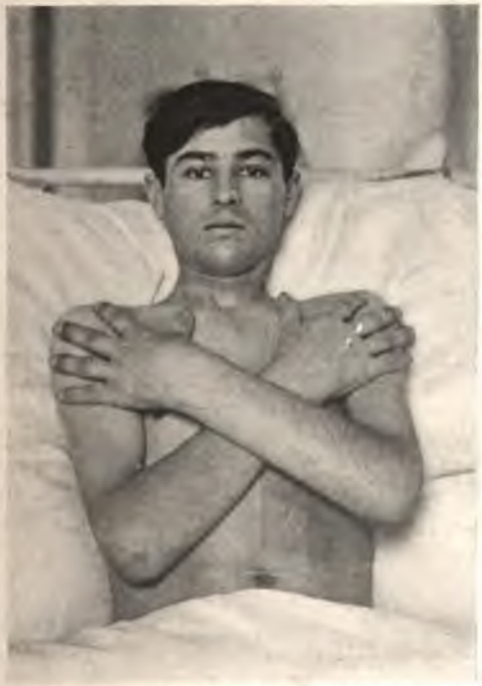


FIG. 3.—Pulmonary osteo-arthropathy of the hands and forearms in a case of sarcoma of the lung.

smaller, the thymus is present, the bones are more cartilaginous (see p. 138).

The Skin.—(*a*) The *color* (pallor, cyanosis, jaundice, pigmentation, mottling, etc.); (*b*) the *character* (texture, moisture, edema, eruptions, gloss, subcutaneous fat, wasting, distended blood-vessels).

The Muscles.—Development, wasting, tremors, symmetry.

The Face.—Intelligence, expression, symmetry, spasm, paralysis, edema, myxedema.

The Hair.—Dryness, sparsity, distribution, dyes, parasites, local discoloration, the presence of vermin.

The Eyes.—Prominence of the eyeballs, the pupils (size, color symmetry, equality, reaction to light, etc.), conjunctiva (color, ecchymosis, discharges), cornea (transparency, arcus senilis, leucoma).

The Mouth.—Teeth, gums, tongue, pharynx, tonsils, lips (pyorrhea, cyanosis, herpes, ulcerations, moisture, deposits, drooping, rhagades).

The Ears.—Shape, discharges, tophi, scars.

The Nose.—Discharges, obstruction, motion of the nostrils (dyspnea), dilated venules.

The Neck.—Pulsations—arterial and venous, swelling—adenitis, thyroidal enlargement, scars.

The Hands.—Cyanosis, curved or ridged nails, clubbed fingers, joints, deposits (tophi, Heberden's nodes), shape, symmetry, nutrition, capillary pulse (Figs. 1, 2, 3, 329, 330, 331).

The Abdomen.—Shape, distention, varicosities, asymmetry, pulsation, edema, eruptions.

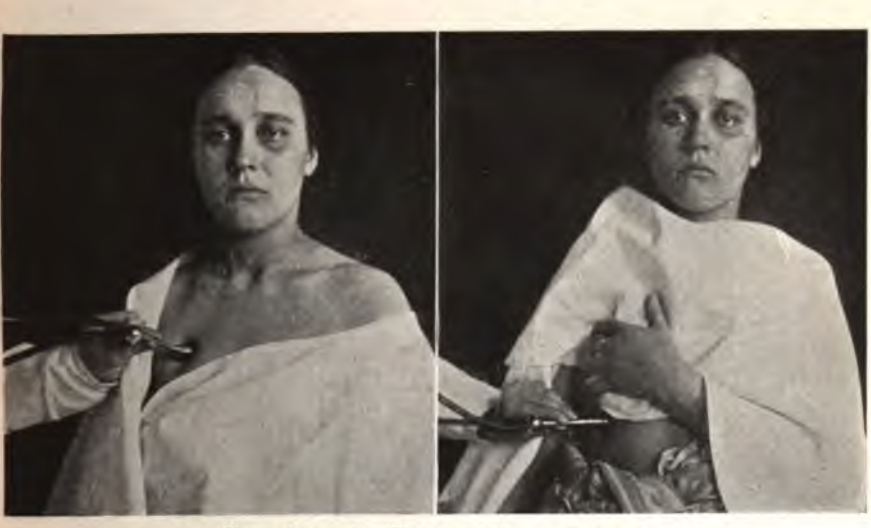
The Legs and Feet.—Edema, clubbing of the toes, deformities, varicosities, cyanosis, scars, pigmentation.

INSPECTION OF THE CHEST

This method of physical examination is too frequently omitted, or made so hastily and cursorily that little or no information is obtained. Inspection, properly done, yields more valuable information than any other procedure at our disposal, with the exception of auscultation; and furthermore, it has this to commend it, namely, that no special training is required, and the beginner, providing he is taught to use his eyes intelligently, is as capable of seeing defects as the experienced observer. This is in marked contrast to the training necessary to educate the ear to differentiate sounds, particularly those produced by percussion, the latter method often requiring years of practice. Inspection, on the other hand, requires no special technique; the only requirement is that one should keep in mind constantly that *every abnormality, however slight it may appear, is worthy of consideration.*

One who has been taught to make a proper inspection can, in many instances, come to a fairly definite conclusion from this procedure alone. Since inspection requires no special training, it is particularly valuable to the student, and to those who see chest cases incidentally, and not constantly.

In order that inspection should yield the best results, it is absolutely essential that the patient be *stripped to the waist.* "The unpleasantness and inconvenience to a patient of undressing for this purpose, the time occupied in so doing, the trouble it gives, and a sense of delicacy in females" are no longer to be considered the serious obstacles Laennec believed them to be. An examination of a chest which has not been entirely exposed is in the vast majority of instances worse than no examination at all. In regard to women it can be safely asserted that no difficulty will be encountered if the importance of the procedure is explained and they are not unnecessarily exposed. For some years we have used the following method. A piece of linen or fine muslin a yard square is slit from one corner to the center and the free edges hemmed (see Figs. 4 and 5). This is thrown over the shoulders. In examining the anterior aspect of the chest the cape is loosened over the shoulders. When the area below the breasts is examined the cape still affords protection. In examining the back, the cape may be pushed up exposing the entire back as no objection is ever offered to this. As the capes are inexpensive a number can be kept on hand and a fresh one used for each patient.



FIGS. 4 AND 5.—The examining cape in use.

It must be borne in mind that a patient stripped to the waist should not be subjected to the discomfort of a cold examining room.

The next requisite is that the patient shall be so placed that the light falls directly on the parts under inspection. In comparing the two sides of the chest, the illumination must not come from one side as errors may occur if one-half of the chest is less well lighted than the other. The chest should be inspected not only from the anterior and posterior aspects but also in profile; the latter method is of value in estimating the depth of the chest and also in determining the presence or absence of pulsation. In addition the chest should be inspected from above downward. This is done by the examiner standing behind the patient and looking down over the shoulders.

The Posture of the Patient.—As to the posture of the patient, the sitting position is the one of choice. The patient should be instructed to assume a natural posture and not, on the one hand, to sit too rigidly, or, on the other, to assume a slouching position. The standing position may be selected if the examiner prefers it, but it is not as convenient and if the examination takes much time is tiring both for the patient and the examiner.

Inspection of the chest in patients who are confined to bed and acutely ill is never as satisfactory as in those who can sit or stand up. Furthermore, only the anterior aspect of the chest, as a rule, is available for the method. Another difficulty is that in private houses the light frequently comes from one side only, so that half of the chest is in a shadow which seriously interferes with a good view. In very ill patients this method of physical examination, in common with the other procedures, suffers from a lack of thoroughness which is often unavoidable. If the examination of the patient in the recumbent attitude is unavoidable, care should be taken to see that the body rests on an even plane; otherwise the results may be affected very materially (see p. 202).

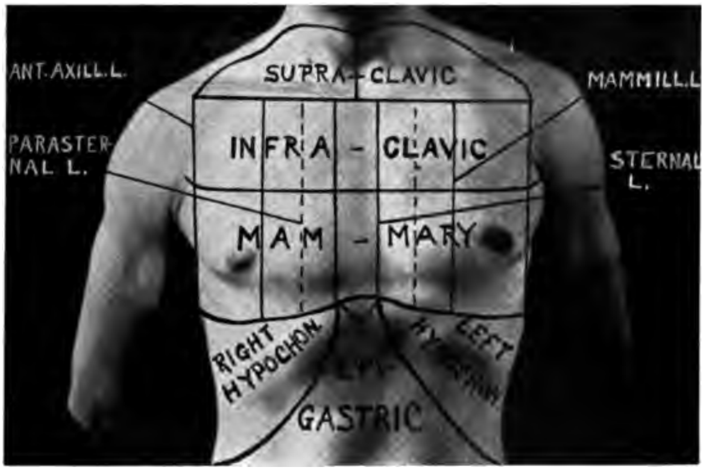


FIG. 6.—The topography of the chest anteriorly. For purposes of description the chest is divided into certain regions which are shown in this and in the following figure.

To fix a standard of what constitutes a normal chest which shall serve as a criterion by which to estimate either the existence of or the degree, of abnormal variations, is not possible. Individuals entirely free from thoracic disease present the greatest variations in the conformation of their chests.

The Conformation of the Normal Chest.—Providing that the chest does not present some one of the recognized deformities, it is assumed to be normal if it is symmetrical, not only generally but in its different parts. The shoulders should be on the same level and the line from the neck to the point of the shoulder slightly convex. In men the clavicles are usually more or less prominent and the supraclavicular spaces a little depressed. In women the clavicles are not uncommonly hidden by adipose tissue and there are no depressions above the clavicles. Beneath the clavicles the chest wall is slightly convex. The intercostal spaces are cartilage

slightly below the surface unless the individual is well covered with fat. Owing to the progressively increasing obliquity of the ribs from behind forward the intercostal interspaces are broader in front than behind. In the majority of individuals a projection of the sternum is visible at the level of the second costal caertilags. This projection or angle is of variable degree and is formed by the articulation of the upper and middle portions of the sternum. It is known as the Angulus Sterni or *Angle of Louis*. In certain thoracic conditions, particularly emphysema, the bulging forward of the upper ribs tends to accentuate this angle. The lower part of the sternum just above the ensiform cartilage is normally slightly depressed.

Viewed from behind the angles of the scapula should be on the same level (corresponding to the spine of the eighth dorsal vertebra) and closely approximated to the chest wall.

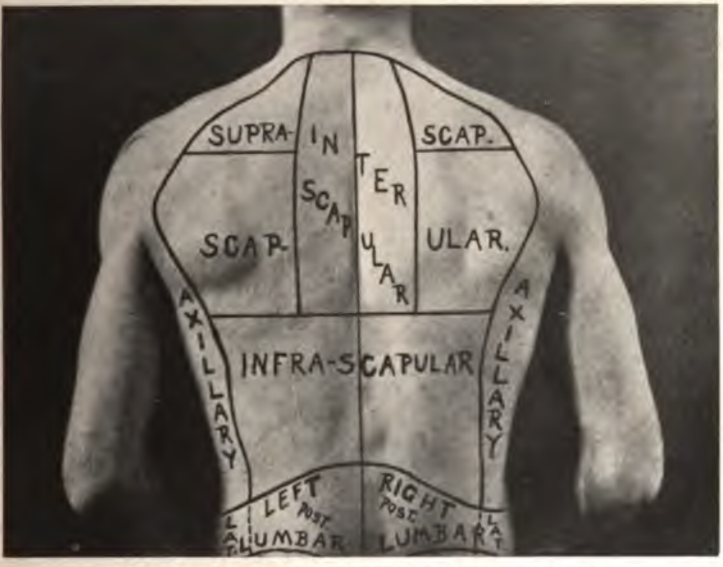


FIG. 7.—The topography of the chest posteriorly.

The spine should be straight and slightly concave from above downward. Slight deviations of the spine are not uncommon, however, and may or may not be an indication of thoracic disease. They are frequently due to faulty posture.

The *points especially to be noted in inspecting the thorax* are: the size and development, the contour and symmetry, the mobility or degree of expansion, the type of breathing, the rate of respiration, the degree of the subcostal angle, local bulging or pulsation, and the prominence of the clavicles.

The Size of the Chest.—The development of the chest depends to a considerable degree upon the general health and activity of the individual. Hence, large, deep, well-muscled thoraces are found in robust, physically active men. Small flat chests are seen as the result of early disease necessitating long periods spent in bed; rachitis, and nasal obstruction (adenoids) are also common causes. Lack of thoracic development or chest deformity in early life is chiefly due to these causes. In adult life abnormalities generally result from tuberculosis, pleuritis or emphysema.

The Contour and Symmetry of the Chest.—It is especially important that the two sides of the chest be compared with each other. There is of course no absolute normal standard, but merely a variable range of the normal. Disease of the chest is so frequently unilateral that by choosing the "better" side we are enabled to estimate a given individual's normal. Asymmetry of the thorax is often due to abnormal curvature of the spine—scoliosis, kyphosis, lordosis—which, when present to a marked degree, often renders examination by auscultation and percussion very difficult (Figs. 17, 26 and 36). Inequality in the size or expansion of the two sides of the chest may be measured by sewing two tape measures together, placing the zero point over the spine and bringing the free ends together, horizontally at the sternum.

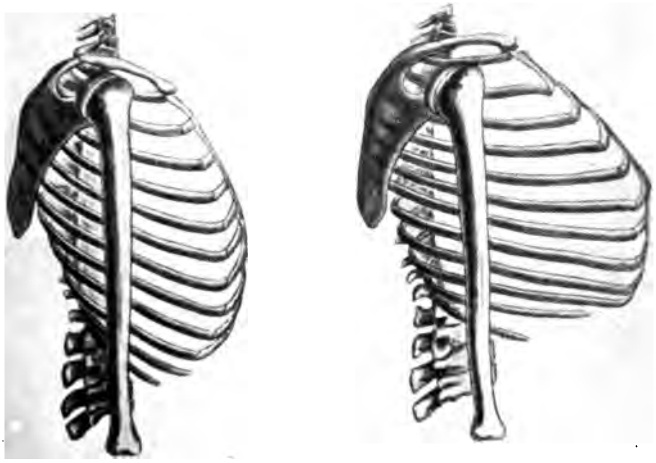


Fig. 8.—Position of the bony thorax during inspiration and expiration. The emphysematous chest is practically that of a chronic, immobile, forced inspiration. (*After Barth.*)

At *birth* the chest is cylindrical (Fig. 19); this form gradually develops, beginning at the second year, into the elliptic shape of adult life (Fig. 20), to revert again during old age to more or less the circular contour of childhood (Figs. 25 and 33). The exact contour can be accurately determined by means of the *cyrtometer*—a band of lead, hinged in the middle, which is firmly applied and moulded to the chest, the contour of which it afterward maintains.

The Mobility of the Chest.—Chest expansion, the difference in circumference between forced inspiration and expiration is in normal men, about 2 inches. Much greater degrees of mobility are found in individuals accustomed to severe physical exercise. Chest expansion can be greatly developed during adolescence by practice; it is medically of minor importance (Fig. 8).

Inequality of expansion has great significance. The main point to be determined is whether both sides of the chest expand equally. The most important cause of unilateral diminished or delayed expansion is tuberculosis of the lungs or pleura, but such a condition also results from one-sided pneumonia, pleuritis (pain), or pleural effusion, pneumothorax, etc. The last two conditions may also alter the shape of the intercostal spaces.

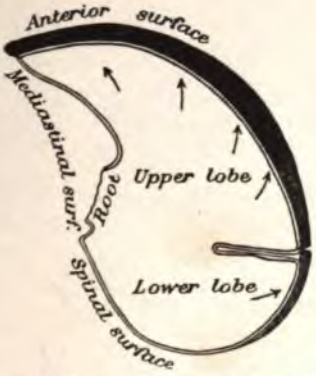
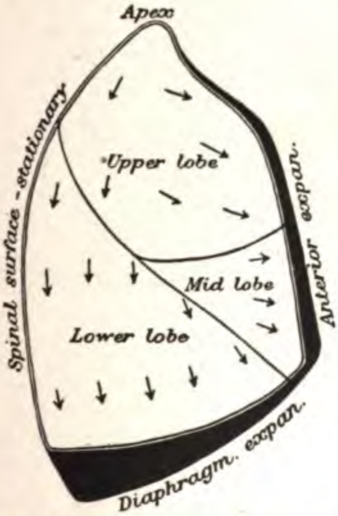


FIG. 9.—Cross section of the right lung showing the direction of expansion. (*Keith.*)



—Right lung from the side showing the direction of expansion. (*Keith.*)

cases, inequality is generally due to local adhesions and con-
the subjacent tissues. Unequal expansion may rarely be
tis, hemiplegia, and muscular atrophy. Diminution or delay
nsion can be best detected by standing behind the seated
ooking downward over his shoulders.
leal regarding pulmonary function can be learned by in-

spection. In order to determine local inequalities of movement over the upper lobes Hoover recommends the following procedure.

The examiner standing on the right of the recumbent patient places the tip of the ring finger of the left hand upon the second rib in the midclavicular line; the tip of the middle finger on the third rib midway between the anterior axillary and the midclavicular lines; and the index finger on the fourth rib in the anterior axillary line. If the patient breathes deeply and somewhat rapidly, normally the relative degree of motion should increase progressively from the first to the third rib. In other words normal expansion is undulatory. If there is diminished ventilation the three ribs will move in unison.

Inspiratory narrowing of the subcostal angle and retraction of the whole of both costal borders is noted as an evidence of sub-ventilation in emphysema, which may appear in association with an aggravation of symptoms such as dyspnea and cyanosis, and increased carbon dioxid concentration in the alveolar air, although percussion may fail to show any change in the position of the pulmonary margins. Such observations may enable us to differentiate an access of dyspnea due to emphysema from that due to cardiac weakness, for in the latter instance although we may find subcostal inspiratory narrowing, we will not find inspiratory retraction of the whole costal margin (Hoover).

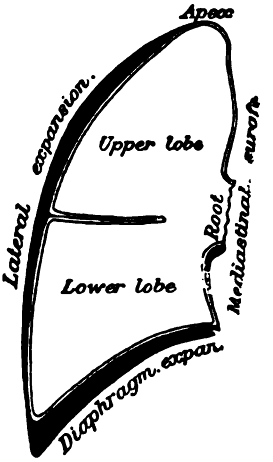


FIG. 11.—Vertical section of the right lung showing the direction of expansion. (*Keith.*)

Unilateral variations may also aid us diagnostically. Thus with left ventricular hypertrophy the median portion of the left costal margin will be retracted or move less in an outward direction during inspiration than the corresponding area of the right chest. In cases of mitral stenosis, myocardial disease, and enlargement of the pericardial sac there will be symmetrical narrowing or constraint of the subcostal angle during inspiration, if the anterolateral portion of the diaphragm is depressed beyond the critical point by the enlarged heart or pericardium.

THE VITAL CAPACITY OF THE LUNGS

The Vital Capacity of the Lungs.—The amount of air which can be expired after a forced inspiration varies in normal individuals with sex and height as well as with muscular development. It depends largely upon the latter factor and upon proper breathing. It can therefore be increased, especially in youth, by training. Low values mean simply poor development and improper breathing, and are of practically no value in the diagnosis of pulmonary lesions such as tuberculosis. In heart disease, however, the pulmonary "vital capacity" corresponds more or less to cardiac efficiency. The degree to which it is reduced below the normal standard corresponds closely to the tendency to dyspnea. Thus patients with marked decomposition show values of only 40 per cent. or less of the normal, while patients with well compensated lesions, who have no greater dyspnea on exertion than normal persons yield values of about 90 per cent. of the normal.[1]

The Type of Breathing.—In men respiration is mainly diaphragmatic, in women, costal.

> "By study of the living thorax in health and disease we learn—That the diaphragm is the great means of inspiration: That, in quiet breathing, the chief use of the intercostal muscles is to maintain the position of the ribs (or the expansion of the chest) during the descent of the diaphragm: That, when the descent of the diaphragm is hindered, or when inspiration becomes more laboured than natural, the intercostals contract more strongly, so as to dilate the chest by raising the ribs: That, when inspiration becomes as forcible as possible, other muscles, which act by raising the collar bones and first ribs, come into play, namely, the sternomastoids, scaleni, omohyoids, and upper part of the trapezii: That quiet expiration is due to the cessation of all muscular contraction: That forced expiration is performed by means of the abdominal muscles (especially the recti), the latissimi dorsi, and lower part of the trapezii. Add these corollaries: That the diaphragm and intercostals are antagonist, although they concur to produce one and the same result: That forced inspiration tells upon the upper chest and true ribs: That forced expiration tells upon the lower chest and false ribs" (Gee).

"Whether respiration be mainly costal or mainly abdominal depends on the relative part taken in the act by the ribs and the diaphragm." The better developed the abdominal muscles are, the more easily can the diaphragm elevate the ribs, and the more "thoracive" the type of respiration. It also depends on "the order in which different parts of the body wall come into action. If the wave begins in the abdomen and passes upward, the type is abdominal; if it begins above and passes down, the type is costal" (Hutchison).

The types of breathing are often modified by disease; thus the pain of an acute pleuritis diminishes expansion especially on the affected side. Peritoneal pain diminishes downward movement of the diaphragm and produces relatively more costal breathing.

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[1] The vital capacity of the lungs is measured by means of the *spirometer*. *Normal values* are: Men; Height 159.5–173.5 cm., 4000 c.c.; from 173.5–182.5 cm., 4800 c.c.; 182.5 and above, 5.100 c.c. *Women:* Height from 154.5–162 cm., 2825 cc.; from 162–167 cm., 3050 c.c.; 167 and above, 3275 c.c. (McClure, C. W. & Peabody, F. W.: Relation of Vital Capacity of Lungs to Clinical Condition of Patients with Heart isease. *J. A. M. A.*, Dec. 8, 1917, lxix, 1954.)

The Rate of Respiration.—The rate of respiration in adult man is from 16 to 20 per minute. In healthy adults breathing is rhythmic as long as they are unconscious of it. In infants, in whom the function is imperfectly established, it is often irregular. To insure accuracy the patient should never know that we are observing the manner and the rate of his breathing. The respiration and the pulse normally bear the relation of about 1 to 4.

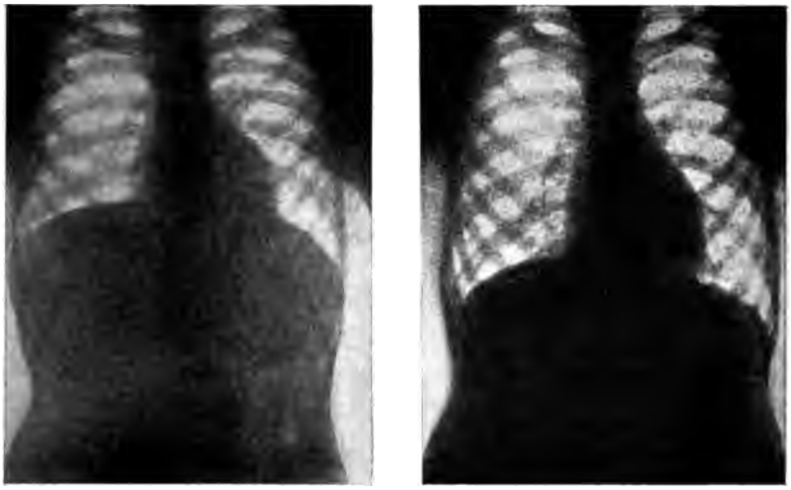

FIG. 12.—Radiogram showing the position of the diaphragm during expiration and inspiration. Note the effect upon the position and shape of the heart.

A constantly *increased rate of respiration* generally indicates disease of the heart or lungs, but disease of the peritoneum and hysteria may also cause rapidity. A *decreased respiratory rate* occurs in sleep (25 per cent. slower), in obstruction to the air passages and in opium poisoning.

NORMAL RATIO BETWEEN AGE, PULSE, INSPIRATION AND TEMPERATURE

		Birth to 2 years	2 to 5 years	5 to 9 years	9 to 12 years	Adult
Pulse rate		122	114	103	89	76
Respiratory rate. T. 98°		30	26	25	24	17
T. 102°	Respiratory	43	35	30	29	27
	Pulse	141	135	128	117	106
T. 105°	Respiratory	50	44	37	31	34
	Pulse	149	161	136	136	136

The Subcostal Angle.—By this is meant the angle at which the ribs meet at the ensiform cartilage. It is important in classifying the type of chest with which we have to deal. It is narrow or acute in the long, flat chest; and broad or obtuse in the cylindrical or barrel-shaped thorax (Fig. 13, also p. 26).

Local Bulging or Pulsation.—Local prominence of the chest is often due to rachitis in childhood and to spinal curvature. The precordium (that part of the chest which overlies the heart) is often normally prominent, although much greater degrees of bulging are seen when the heart is hypertrophied (Fig. 15, 217), especially in children. The most important pathologic prominence is that seen at the base of the heart in aneurism of the aorta (Fig. 16). Occasionally disease of the ribs and empyema cause local swelling. In emaciated subjects pulsation of the subclavian arteries may be visible. Rhythmicity practically indicates that a pulsation is directly connected with the circulatory apparatus. Pulsating empyemata are very rare.

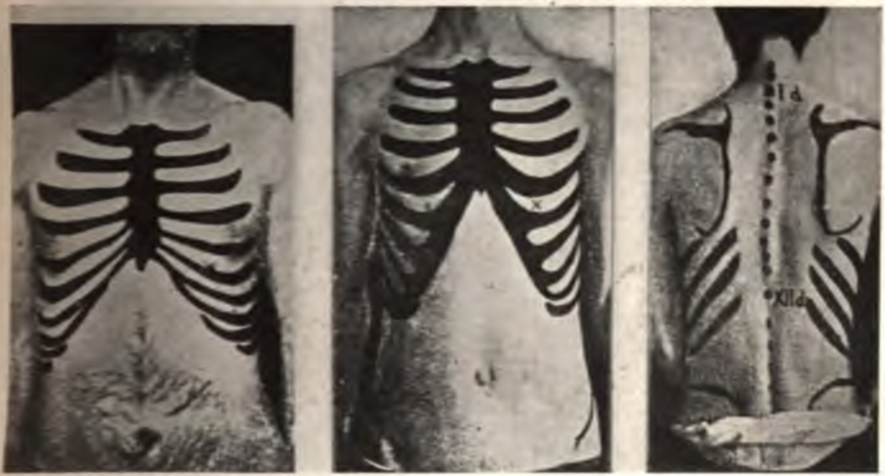

FIG. 13.—Illustrating the angle of the ribs in the barrel-shaped and in the long, flat chest. (*Fowler and Godlee.*)

Prominence of the Clavicles.—This depends largely upon the shape of the chest. It is marked in thoraces conforming to the "long, flat" type and hence is common in enteroptotic individuals. It is often seen in tuberculosis of the pulmonary apices, and when unilateral, may be regarded as an index of the degree to which the subjacent tissues have sunken in or have been retracted (Fig. 30).

The Diaphragmatic Shadow.—(*Phrenic wave, Litten's phenomenon*) (Fig. 18).—If a moderately thin person, lying on a bed with the chest exposed and with the feet pointing directly toward the window, takes slow, deep, "abdominal" inspirations, a shadow will be seen to move down the lateral aspect of the chest wall, between the sixth and the ninth ribs. This shadow is caused by the fact that during inspiration the diaphragm "peels off" from the inner chest wall and by means of the negative pressure thus produced, causes the soft tissues to fall inward. These alterations of pressure correspond with in- and expiration and create the impression of a shadow moving down the chest wall. By the mobility of the shadow and by the extent of its excursion, we are enabled to determine the degree of diaphragmatic motion and its equality on the two sides of the body (Fig. 18).

Local adhesions, a pneumonia, or a pleural effusion would diminish or inhibit the shadow. A subdiaphragmatic abscess would, on the

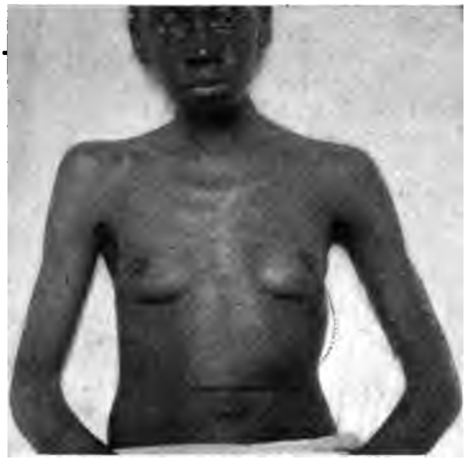

FIG. 14.—Bulging of the left lower chest, which pulsated synchronously with the heart even after aspiration of the pleura, due to an aneurism of the left ventricle. The dotted area represents approximately the degree of pulsating excursion. (*Patient of Dr. J. N. Henry.*)

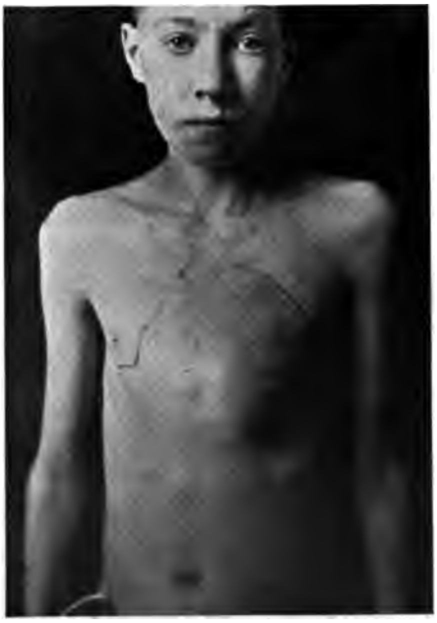

FIG. 15.—Bulging of the precordium due to an hypertrophied heart (mitral obstruction) in a boy of 14 years. Note the poor nutrition and underdevelopment often caused by cardiac lesions in childhood

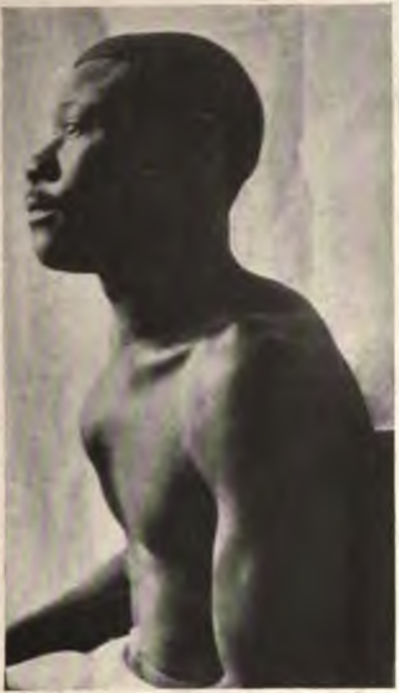

FIG. 16.—Deformity of the chest due to thoracic aneuri

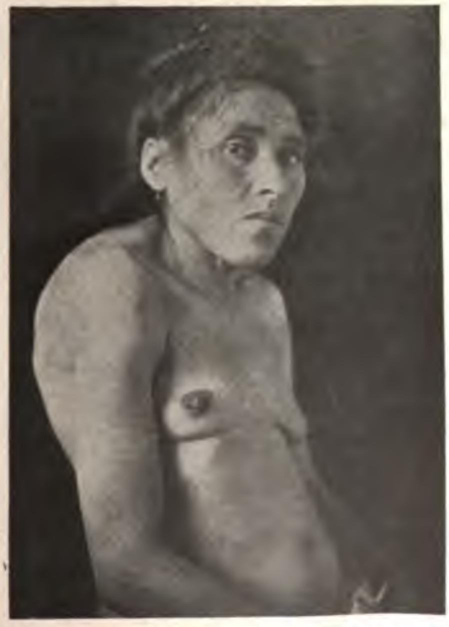

FIG. 17.—Deformity of the chest resulting from kypho-scoliosis. Abnc
must be construed with great caution in these cases. (See Fig

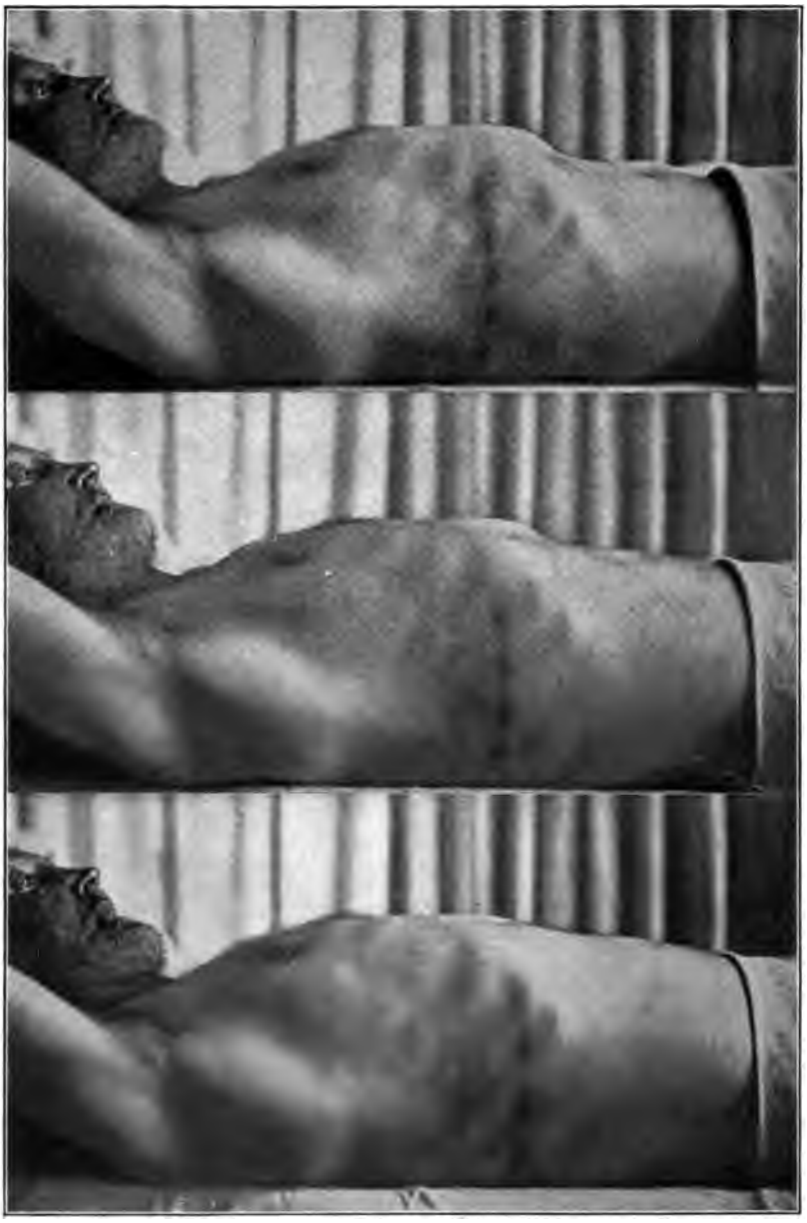

FIG. 18.—Diaphragm phenomenon (Litten's sign), from a patient with fibroid phthisis of left lung (New York City Hospital). The linear shadow has been emphasized in the reproduction of the photographs.

1. *Full Expiration.*—Note the height of the shadow and the slight concavity of the abdomen corresponding to the respiratory phase.
2. *Medium Inspiration.*—Note the descent of the linear shadow and the slight change of contour of abdomen corresponding to the respiratory phase.
3. *Deep Inspiration.*—Note the further descent of the linear shadow and the rigid abdomen corresponding to the respiratory phase.

Although the artist has intensified the shadow in the reproduction, the excursion of the right lung and right side of the diaphragm were so pronounced in this patient that the distance between the shadows in the extreme positions of respiration was greater than has been represented. The patient's left lung was practically useless; hence the abnormal extent of the right lung's excursion. (*Sahli and Potter.*)

other hand, have *per se* no effect on the excursion. The shadow is seen with difficulty in (1) obese subjects, (2) if there is more than one source of light, (3) if costal breathing is practised. The extent of the normal excursion ranges between 2½ and 3½ inches, depending upon the depth of respiration. The observer may stand between window and patient, or may have the patient between himself and the window. The patient should be told to "breathe with his belly."

Diaphragmatic mobility can, of course, be much more accurately determined by means of the fluoroscope but in the absence of such an apparatus the shadow test may be of considerable diagnostic utility. Unilateral immobility or diminished diaphragmatic excursion generally results from pleurisy. Among 83 cases studied by Pryor 53 showed complete immobility, and 17 some restriction of motion, in patients who had previously had pleural effusions.

ABNORMAL THORACIC CONFORMATION

As a result of disease, the shape of the chest often becomes altered from the average normal type. There are, of course, also congenital tendencies which have their influence, but practically the shape of the chest is due to postnatal influences. Freund and others maintain that thoracic development is absolutely determined as regards its ultimate form by the rapidity of growth and time of calcification of the first rib.

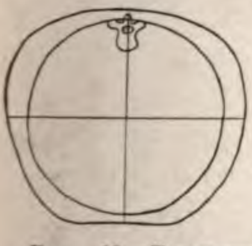

FIG. 19.—Transverse section of an infant's chest. Its shape is cylindrical.

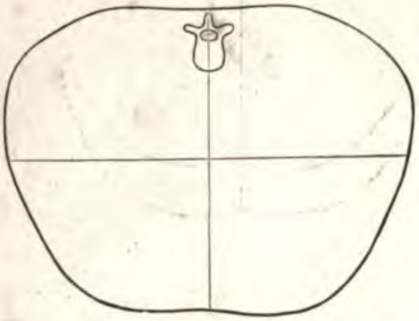

FIG. 20.—An adult's chest, elliptical in shape.

The length of the normal first rib:

	Male	Female
According to Freund is	3.8 cm.	3.1 cm.
According to Hart is	3.6 cm.	3.02 cm.

In flat-chested (phthisical) individuals it measures from 3 down to 2.2 cm. This makes the upper chest much narrower and since the rib is also more sloping, makes the whole antero-posterior thoracic diameter small. The first costo-sternal articulation is frequently ossified. In thoraces having this conformation the pulmonary apices are more slender and respiratively less mobile. It is to these factors that the predisposition to tuberculosis is supposed to be in part due.

Pathologic deformities of the chest may be *classified* among the following types:

I. The Rachitic Chest.—The rickety (rachitic) chest (due to unduly soft bones during its development) is typically characterized by (1) beading

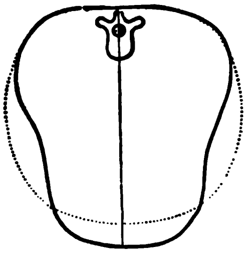

Fig. 21.—The rachitic chest.

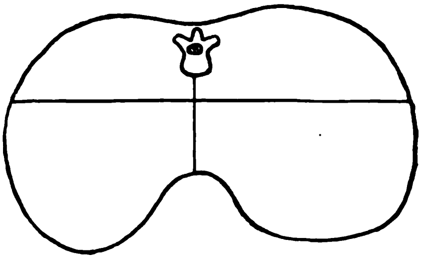

Fig. 22.—The funnel-breasted chest. (Compare Fig. 30.)

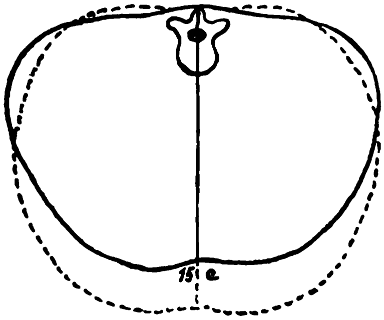

Fig. 23.—The flat chest.

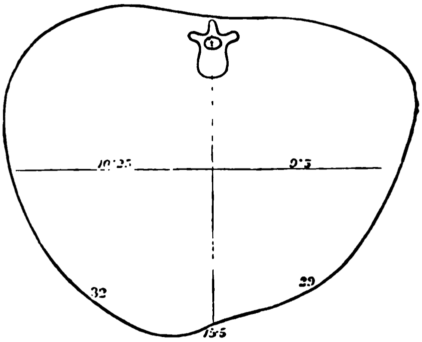

Fig. 24.—Unilateral retraction of the chest due to pulmonary fibrosis.

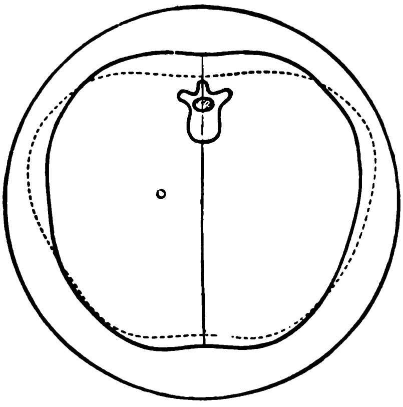

Fig. 25.—The emphysematous chest. (Compare Fig. 33.)

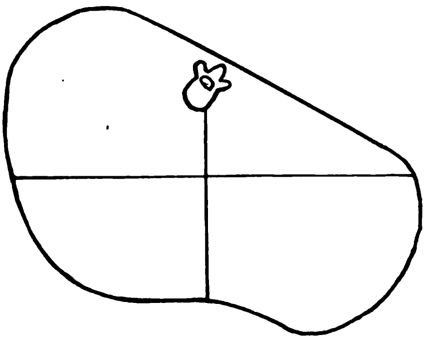

Fig. 26.—The chest in scoliosis. (Compare Fig. 17.)

of the costo-chondral junctions (rachitic rosary), (2) a transverse furrow corresponding to the attachment of the diaphragm (Harrison's groove), (3) prominence of the sternum (pigeon breast), (4) a longitudinal groove parallel to the sternum extending to the costal margin.

During infancy respiration is mainly abdominal, because the thorax is already cylindrical and admits of but little further outward expansion. The negative intrathoracic pressure occasioned by the descent of the diaphragm exerts its effects mainly on the lower ribs. The atmospheric pressure causes the most yielding portion of the thorax to cave in (costo-chondral articulations) and thus a depressed groove, parallel to the sternum is formed. Such deformities may result from normal breathing, but are much enhanced in case of inspiratory dyspnea. Forced inspiration, which overexpands the upper chest, causes a forward protrusion of the sternum which results in "*pigeon breast*" (Fig. 29). This may be unequal in degree on the two sides as the result of scoliosis. The chest is usually shortened, and the costal angle acute. Occasionally there may be a depression of the sternum extending from the middle of the gladiolus to the ensiform cartilage (*funnel breast,* trichter brust, pectus excavatum), (Figs. 28 and 30). Although this deformity is usually congenital, it may be occupational (schuster brust, cobbler's breast) due

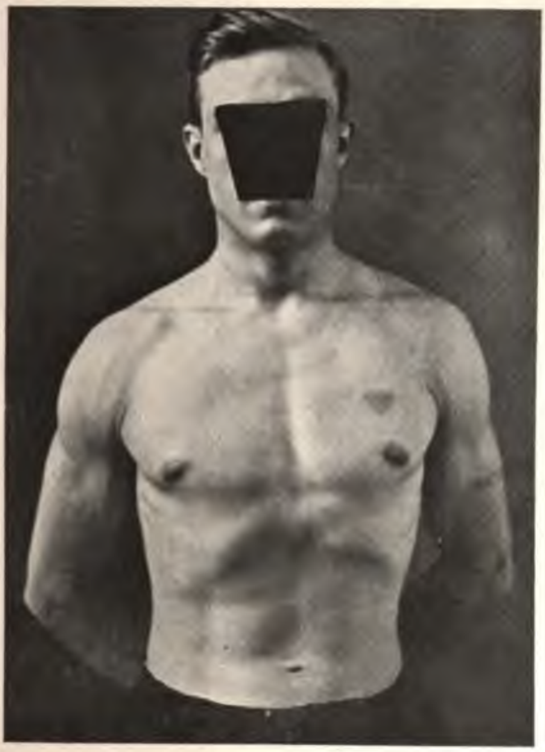

FIG. 27.—Normal chest with excellent conformation and muscular development. The pigmented area above the left nipple is the result of a recent blister.

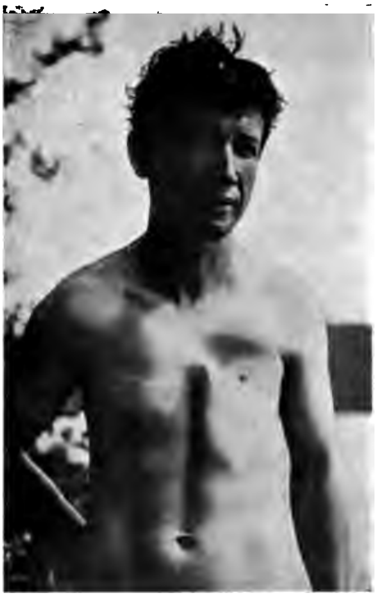

FIG. 28.—A rachitic deformity of the chest which produced a very pronounced dextrocardia.

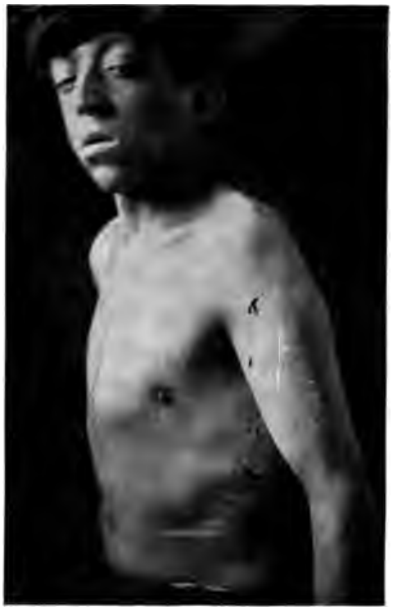

FIG. 29.—Pigeon breast in a cretinoid dwarf, aged 23 years.

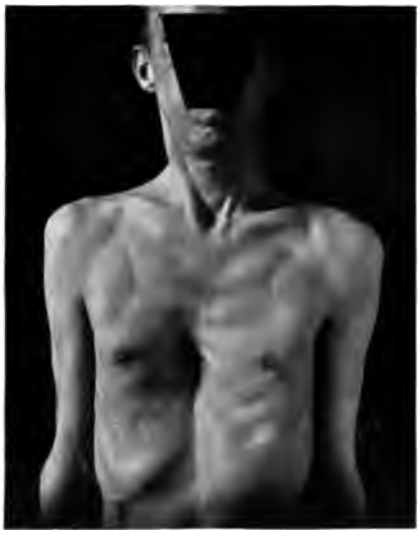

FIG. 30.—Funnel breast due to rachitis, in a patient who ultimately developed pulmonary tuberculosis. (*Patient of Dr. Ward Brinton.*) (Compare Fig. 32.)

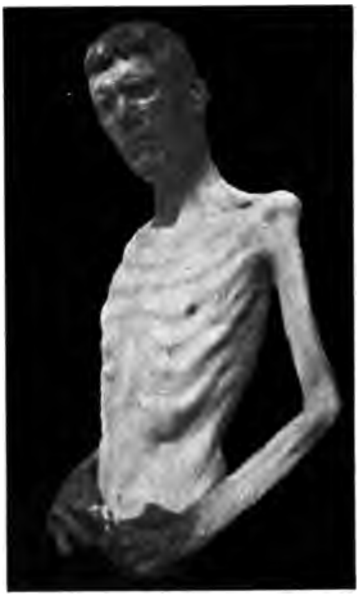

FIG. 31.—Long, flat chest with marked retraction and emaciation, in a case of chronic pulmonary tuberculosis. (*Courtesy of Dr. Frescoln.*)

to external pressure from tools of trade. A transverse section of a pigeon breast is triangular in form (Fig. 21).

II. The Long Flat Chest.—The flat (phthisinoid, paralytic) chest is commonly met with in pulmonary tuberculosis, and while apparently predisposing to, is often the result of, this disease. It is a chest of diminished capacity and functionation. The thorax is elongated; the normal elliptical shape is flattened (antero-posterior diameter relatively decreased), the subcostal angle is acute, and the obliquity of the ribs increased. In association we often see faulty posture (stoop shoulders, pot bellies), poor expansion, a long neck with a prominent larynx and the head carried forward. Increased obliquity of the ribs makes the scapulæ prominent (winged) and in such cases we speak of the "pterygoid" or "alar" *chest*, especially if the lateral diameter be small. The clavicles are prominent, the supra- and infraclavicular fossæ exaggerated and the interspaces wide. Not infrequently rachitic deformities, and especially unilateral deformities due to pulmonary fibrosis are superadded (Figs. 30, 31, and 32).

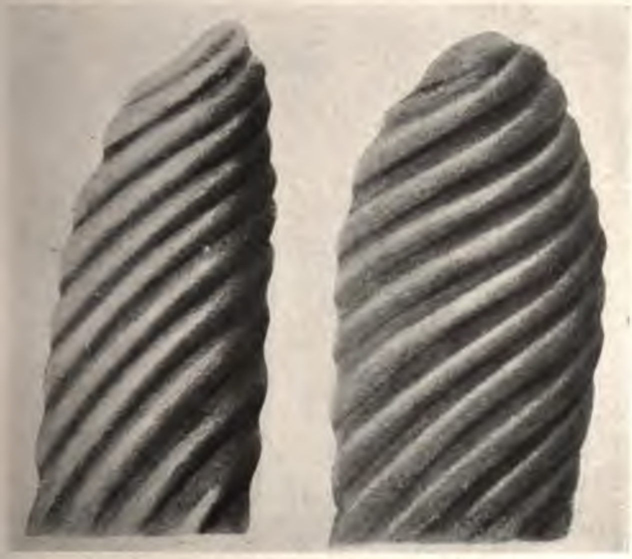

FIG. 32.—Plaster cast of a long flat, and a normal chest. (*Houseman.*)

III. The Barrel-shaped Chest.—In the emphysematous (barrel-shaped) chest the normal elliptical form tends to become cylindrical (the cylinder has a greater cubic capacity). The ribs are elevated and everted (position of forced inspiration); as a result of this the costal angle enlarges, the lower ribs flare upward, and the sternum is arched; while Louis' angle becomes prominent.

These changes are bilateral and result from enlarged thoracic contents—enlarged lungs, emphysema. If the spine is also involved it becomes bent backward, the shoulders are thrown forward and the back is rounded (Figs. 8, 33, 34 and 35).

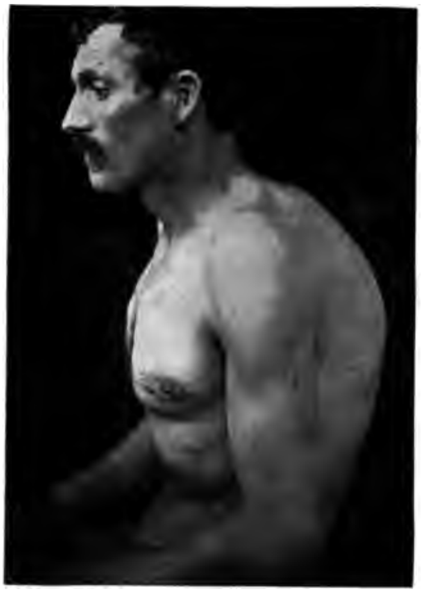

FIG. 33.—Barrel-shaped chest, a case of long-standing pulmonary emphysema. (Compare Fig. 25.)

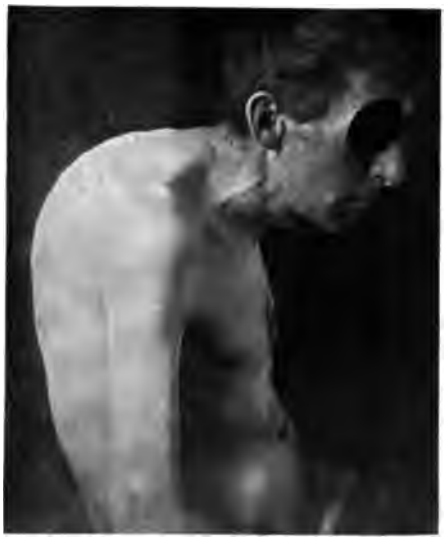

FIG. 34.—Emphysematous chest with marked kyphosis. (*Patient of Dr. Ward Brinton.*)

This type of chest is the result of prolonged dyspnea—asthma, emphysema—owing to which the accessory muscles of respiration (sternomastoids, serrati postici superiores, serrati antici majores, trapezii, rhomboids, levatores scapulæ, and the pectoral muscles) hypertrophy. The neck becomes thick and short, the thorax being drawn upward. Inasmuch as the expiratory muscles are also called upon for increased effort the abdominal muscles, the quadrati lumbori, and serratus anticus major also become enlarged.

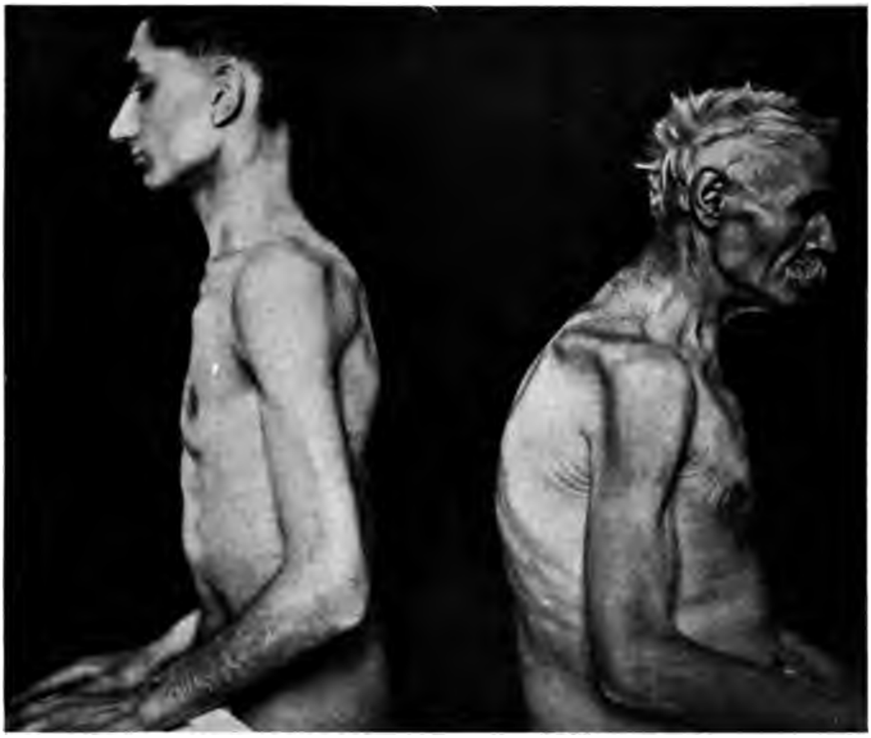

Fig. 35.—Contrasted Types. The young man on the left—a case of advanced pulmonary tuberculosis—has a long, flat chest. The ribs are obliquely placed, the antero-posterior diameter is decreased, expansion is almost nil. The neck is long, the larynx, clavicles and scapulæ prominent.

The old man on the right—a case of pulmonary emphysema—has a barrel shaped chest. The ribs are horizontal, the spine is arched, the antero-posterior diameter is increased, the neck is short, the accessory muscles of respiration are hypertrophied, the jugular veins are distended.

In some cases only the upper part of the chest is involved. The emphysematous chest is in varying degree a normal senile change, it is also seen in individuals whose occupations require habitual stooping, especially if associated with great respiratory demands—laborers, sawyers. The most typical examples occur in sufferers from emphysema especially if associated with asthma. The supraclavicular fossæ are full and may actually bulge during expiration (see Figs. 13, 25, 32, 33).

Mixed Deformities.—Various combinations of the foregoing types may sometimes exist.

There may also be unilateral enlargement, depression or bulging. Such changes are usually due to spinal abnormalities, or to pleural and pulmonary fibrosis.

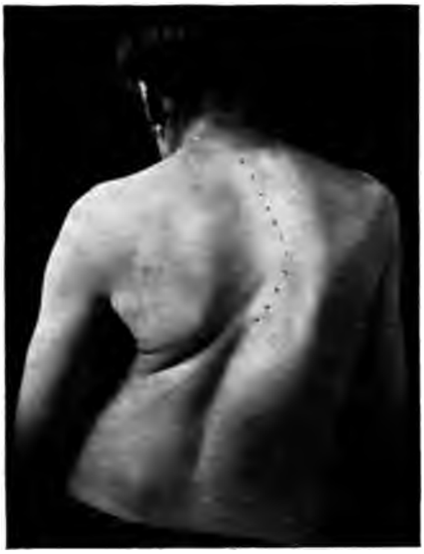

Fig. 36.—Chest deformity resulting from kypho-scoliosis. (See p. 93.) (Compare Figs. 17, 26, 34.)

ABNORMAL RESPIRATION

Dyspnea.—This term is applied to various types of "shortness of breath." It generally means rapid, labored respiration but is also applied to the slow difficult breathing which occurs when the larynx or the trachea is obstructed. Dyspnea may occur in isolation, during either phase of respiration, inspiration or expiration, but usually both acts are affected. It is due to either (1) lack of oxygen, or (2) excess of carbon dioxid in the blood. As a general rule the rate and the depth of respiration bear an inverse ratio toward each other.

Recent investigations[1] have shown that increased *Hydrogen ion concentration* of the blood (excess of CO^2) increases the *depth* of respiration, without essentially altering its rate, whereas *anoxemia* (Oxygen deficiency) produces first, a peculiar *periodicity* of the respiratory rhythm; and secondly, if anoxemia becomes still more marked, very *rapid* and *shallow breathing*.

Inspiratory dyspnea without obstruction simply causes increased thoracic movement; *with obstruction* it causes an over expansion of the upper, and a recession of the lower chest. This in rachitic children produces marked deformities.

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[1] Haldane, Meakins and Priestley: *J. Physiol.*, 1919, lii, 420.

Expiratory dyspnea occurs in asthma, emphysema and edema of the glottis. Expiration is prolonged and laborious, the accessory expiratory muscles are called into activity.

Orthopnea is exaggerated dyspnea, in which the patient is no longer able to breathe in recumbency, but is forced to sit up and frequently add the additional support of his hands, in order to fix the muscles of the shoulder girdle and thus assist the accessory muscles of respiration. It is characteristically seen when the lungs are congested as the result of tricuspid insufficiency, and indicates that the last line of reserve force has been called into action.

Asthma is an intermittent form of dyspnea in which *expiration* is chiefly affected. The latter is often audible at a distance from the patient as a musical wheezing sound. When marked, it is accompanied by cyanosis, a sense of thoracic constriction and of suffocation. It is due to obstruction of the bronchi, either spasmodic or edematous in nature, and is accompanied by the expectoration of tenacious glairy mucus. Asthma is frequently an anaphylactic manifestation.

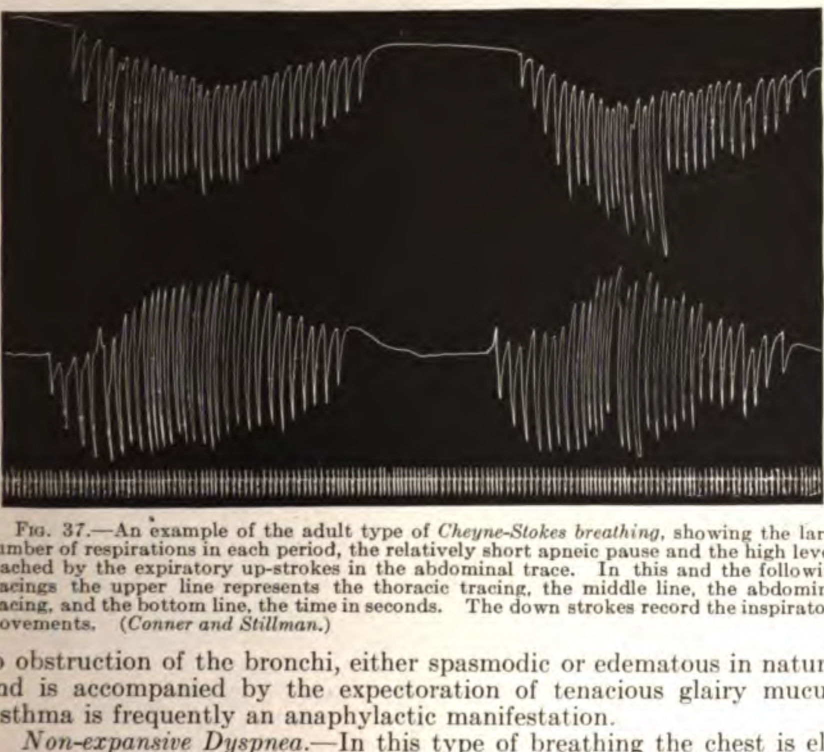

FIG. 37.—An example of the adult type of *Cheyne-Stokes breathing*, showing the large number of respirations in each period, the relatively short apneic pause and the high levels reached by the expiratory up-strokes in the abdominal trace. In this and the following tracings the upper line represents the thoracic tracing, the middle line, the abdominal tracing, and the bottom line, the time in seconds. The down strokes record the inspiratory movements. (*Conner and Stillman.*)

Non-expansive Dyspnea.—In this type of breathing the chest is elevated but does not expand. It occurs if the lung is impermeable to air (pneumothorax), or if the thorax is already distended to the limit of its capacity (large pleural effusions, extreme emphysema) or if expansion is prevented by dense pleural adhesions (pulmonary fibrosis).

Restricted or "catchy" respiration may occur in acute pleuritis or intercostal neuritis as a result of pain.

Stridulous breathing is characterized by a noisy, high-pitched, crowing or whistling expiration. It is due to a spasmodic condition of the vocal cords which occurs in children.

Stertorous breathing may be heard at a distance from the patient. It occurs in comatose and moribund patients, and consists of rattling, snoring or bubbling sounds. Its genesis is akin to that of snoring, since it is often due to vibrations set up by the soft palate, glottis, tongue or vocal cords. Not infrequently, however, it is due to exudate in the trachea or large bronchi, which is thrown into vibration by the act of breathing, as for instance in case of pulmonary edema.

The *death rattle* is a combination of stertorous breathing combined with the rattling caused by the vibration of exudate (serum, pus, blood) in the large bronchi and the trachea.

VISIBLE CHANGES IN RESPIRATORY RHYTHM

Cheyne-Stokes Respiration.—This is characterized by visible irregularity of breathing. The respirations, shallow at first, gradually increase in depth and rapidity, to be followed after gradually diminishing excursions by complete apnea, lasting sometimes nearly 30 seconds (Fig. 37). It *occurs in* the coma of uremia, apoplexy, meningitis, opium poisoning, etc. Blood-pressure is higher during the hyperpneic periods, in cases associated with increased intracranial tension. Cheyne-Stokes respiration is *due to* an obtunded sensibility of the meduall to CO_2 Deep breathing carries off the excess of this substance and the respiratory center remains inactive until another over-accumulation of this gas has taken place. Cheyne-Stokes respiration is of grave, but not necessarily fatal, import. In children, if associated with other suggestive symptoms, it points gravely toward a meningitis.

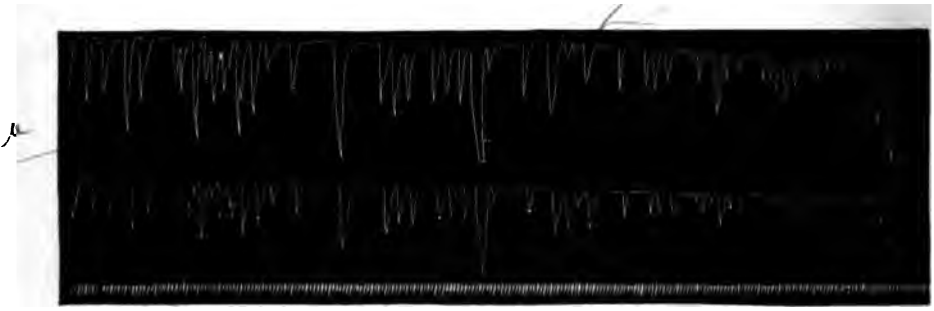

Fig. 38.—Tracing from a child of 8 years, showing typical *Biot's breathing.* Note the pauses which occur at irregular intervals and vary in length, the frequent deep sighs, and the constant irregularity of the respirations in force and rhythm. (*Conner and Stillman.*)

Biot's Breathing.—This type of breathing differs from that just described, in that a series of rapid but equally deep respiratory movements is followed by a sudden apnea. There is no gradual increase and decrease in the depth of respirations (Fig. 38). It may be regarded as almost pathognomonic of *meningitis* (*Conner and Stillman*[1]).

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[1] Conner and Stillman: "A Pneumographic Study of Respiratory Irregularities in Meningitis." *Arch. Int. Med.*, ix, 1912, 203.

CHAPTER II

PALPATION

By palpation we refer to the use of the sense of touch for the determination of the physical character of the tissues. It is employed:

1. To elicit tenderness, or rigidity.
2. To ascertain the position and character of the cardiac impulse, pulsations or thrills, and to feel the pulse (see Chap. XIII).
3. To determine the character of the skin (temperature, moisture, texture, edema).
4. To discover local swelling, induration, softening, etc., of the tissues, especially as regards the lymph nodes.

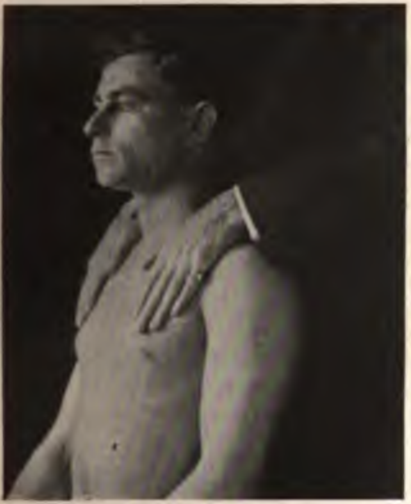

Fig. 39.

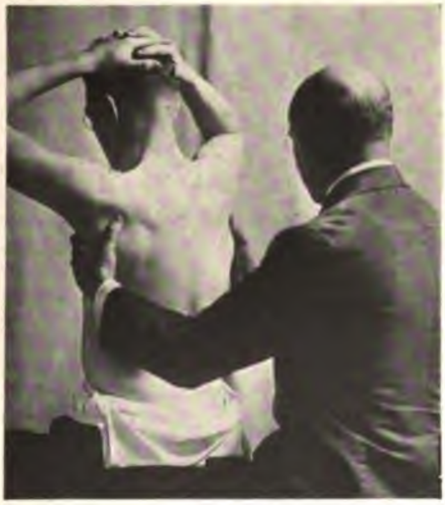

Fig. 40.

Figs. 39.—Palpation to determine expansion and vocal fremitus.
Fig. 40.—Palpation to determine bilateral equality of chest expansion. (*Bethea.*)

5. To estimate the degree and equality of chest expansion.
6. To elicit vocal fremitus.
7. To determine the presence of hepatic or splenic enlargement, pulsation of the liver, etc.

Chest Expansion.—The bilateral equality of chest expansion can often be satisfactorily determined by laying the hands lightly upon the patient's chest during the act of respiration. Some examiners prefer this method to simple inspection.

ZONES OF CUTANEOUS HYPERESTHESIA (HEAD'S AREAS), AND REFLEX PAIN

"The nerves supplying the skin and skeletal muscles have become so educated that any injury to them is accurately located. Such is not the case with nerves of the internal organs. A painful irritation of the viscera finds expression, not necessarily over the site of the organ, but in a painful area of the skin often remote from it. Head has shown that the painful stimulus in the organ travels in a centripetal direction to the posterior part of the cord and there sets up excitation of the nerves which in the same and in the adjoining segments supply the peripheral surfaces with sensation. The pain is referred to the skin because therein the pain sense reaches its highest development."[1]

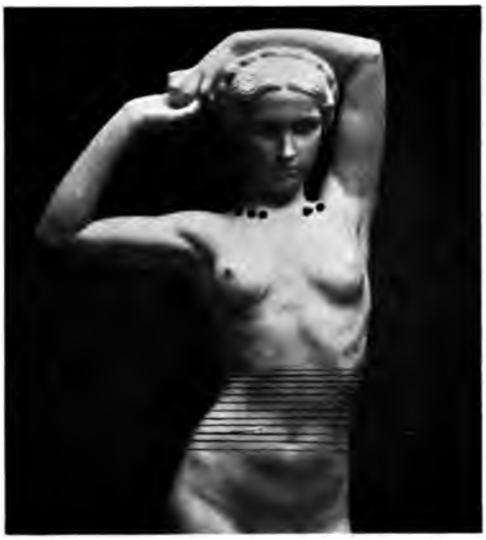

FIG. 41.—Referred pleural pain. The horizontal lines indicate the location of intercostal nerve pain; the dots represent phrenic nerve pain, on the anterior surface of the body.

"Referred pain from the viscera has these characteristics to differentiate it from pain of peripheral origin: (1) It is often remote from the site of irritation. (2) It follows the lines on the skin of the spinal segmentation rather than the course of the peripheral nerves. (3) It is usually associated with cutaneous hyperesthesia and tenderness to pressure. (4) Often the pain fails to involve the whole segmental area of the skin, but finds expression in one or more points of maximal tenderness and spontaneous pain."

Examples.—The pain of biliary colic is felt at the angle of the scapula, that of renal colic in the testicles, while heart pain is referred to the arm or the neck.

Hyperesthesia of the skin is determined by light pressure with a dull object such as the head of a pin, or by slight pinching. Muscular rigidity or spasm which may have a similar genesis is elicited by estimating the degree of muscular tonus by means of our sense of touch.

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[1] CAPPS: *Arch. Int. Med.*, December, 1911.

Cutaneous hyperalgesia was found in only 3 per cent. of all *pulmonary cases* or in 13 per cent. of those who complained of pain. In *diseases of the heart and aorta* the hyperalgias was noted in 7 per cent. of the cases and in 18 per cent. of those who complained of pain.[1]

PLEURAL PAIN

The parietal pleura and outer part of diaphragm receive their nerve supply from the lower six intercostal nerves. Lesions in this region produce pain in the overlying skin, which may be regarded as the result of a peripheral neuritis. Cutaneous hyperesthesia is absent. Irritation of the diaphragm posteriorly produces pain in lower chest, abdomen or lumbar region. This is a referred pain—by way of the seventh to the

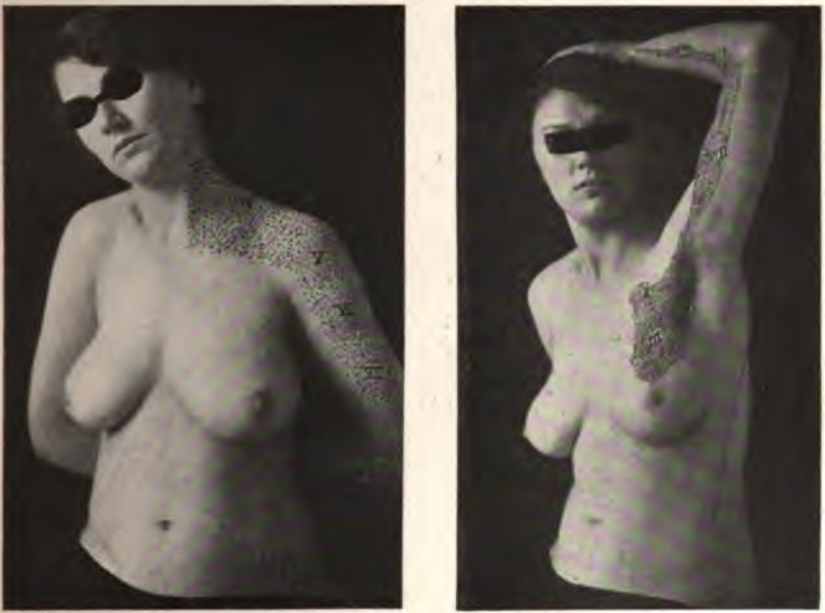

FIG. 42. FIG. 43.

FIGS. 42 AND 43.—Referred cardiac pain. Showing the area of pain and cutaneous hyperesthesia requently met with in angina pectoris. The segmental distribution (third cervical, to the third dorsal) is indicated by the Roman numerals. Other segments may, however, be involved, the sixth dorsal causing epigastric pain, and the upper cervical roots, pain in the neck and back of the head. The characteristic sense of *thoracic constriction* has been explained as resulting from reflex stimulation of the intercostal muscles, and the sense of *impending dissolution* has been likened to the violent stimulation of the nervous system produced by excitation or injury of other viscera, as by a blow on the testicles or over the solar plexus. (*Mackenzie.*)

In pulmonary disease cutaneous hyperalgesia occurred in 3 per cent. of the 460 cases studied by Langstroth and in 13 per cent. of those who complained of pain. In disease of the heart and aorta the hyperalgesia was noted in 7 per cent.; and in 18 per cent. of the cases having pain. As a diagnostic aid the occurrence of Head's hyperalgesic zones is generally disappointing, owing to the large number of areas over which disease of a given viscus may cause sensory changes.

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[1] LANGSTROTH: *Arch. Int. Med.*, 1915, xvi, No. 2.

twelfth dorsal segments. The visceral pleura is devoid of the pain sense. The central diaphragmatic pleura is supplied by the phrenic nerve. Irritation of this region produces pain and tenderness in the neck, especially along the ridge of the trapezius muscle, often with a surrounding zone of hyperesthesia (Fig. 41). This is a true referred pain—by way of the third and fourth spinal segments. The pericardial pleura when irritated may produce similar pain, because its nerve supply is mainly if not entirely phrenic in origin (Capps).

Pleural pain may be referred to the abdominal wall and lack of a careful examination of the lungs has led to unwarranted celiotomies in patients who a day or two later have developed well-marked signs of the pneumonia which was the cause of the original abdominal pain and rigidity. *Vice versa,* although less frequently, abdominal lesions may occasionally produce thoracic pain.

TACTILE OR VOCAL FREMITUS

Fremitus is *the tactile perception of vibrations,* which may be produced by (1) phonation (vocal fremitus), (2) coughing (tussive fremitus), (3) breathing (rhoncal fremitus) produced by exudation into or stenosis of, the air passages.

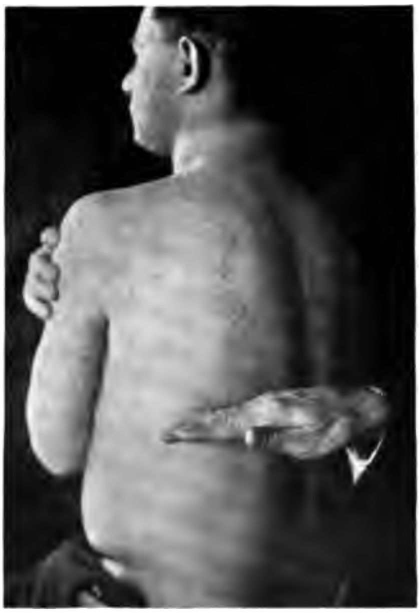

FIG. 44.—Ulnar palpation. The lower pulmonary border can be accurately determined by laying the ulnar surface of the hand and little finger against the chest wall while the patient counts one—one—one—. (See Fig. 50.)

These actions set up vibrations within the bronchi and lungs, which under favorable conditions are accompanied by objectively sensible vibrations of the chest wall, which can be felt when the hand is laid upon it, as a faint vibration or trembling of its surface. The sensation thus obtained has been likened to that derived from a purring cat, but in the case of human fremitus the vibrations are much finer and more rapid.

Vocal fremitus, which only is of practical utility, is elicited by laying the ulnar surface of the hand or fingers upon the chest or in the intercostal spaces, while the patient slowly repeats the numerals, "one—one—one," or, "ninety-nine" in a clear, low tone. Symmetric areas of the chest are then compared in regard to the intensity of the vibrations which are felt. In order to appreciate the mechanism and significance of fremitus we must revert for a moment to that of voice production.

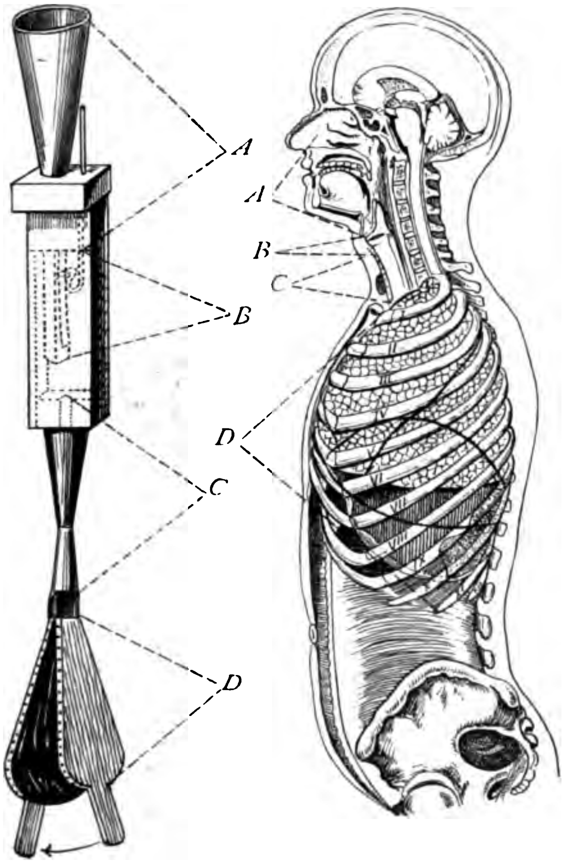

Fig. 45.—Diagram illustrating the similarity in mechanism between voice production in the human body, and sound production in an organ. From above downward the angulated lines indicate respectively: *A*, the resonator; *B*, the reed; *C*, the wind pipe; *D*, the bellows. (*After Barth.*)

The Mechanism of Voice Production.—The larynx is a reed instrument, the pitch of which is determined by the length and tension of the vocal cords. When these are approximated and air is forced upward from the lungs by means of the diaphragm and other respiratory muscles, the cords are thrown into vibration and a sound is generated. The sounds thus produced by the vocal cords and glottis pass upward through a variable resonator—mouth and nares—and are there modified as regards their overtones by the lips, tongue, etc., and thus the vowel sounds originate. A further modification of the overtones by means of the lips, teeth, tongue, etc., results in the consonants of articulate speech. The laryngeal sounds are further reinforced by means of a large resonator—the thorax—which under favorable conditions vibrates in unison with the larynx and, therefore, amplifies the sonorous waves and intensifies the sound, as does a sound box those of the tuning fork. Vocal fremitus is, therefore, not an accidental vibration of the thorax but is a rhythmic vibration due to, and in unison with, the vocal cords. In order *to produce vocal fremitus we must, therefore, have:*

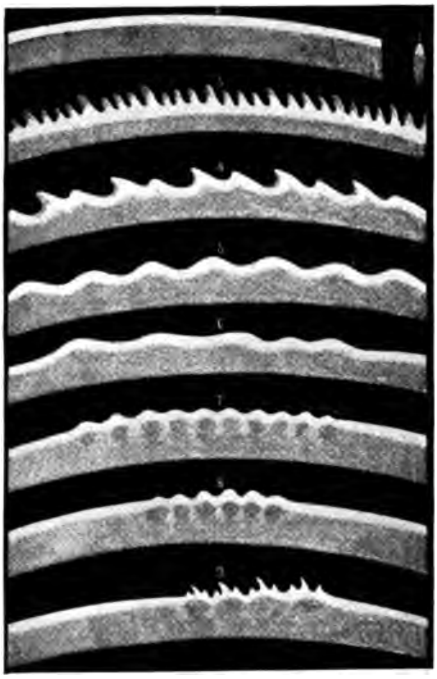

FIG. 46.—Illustration: from Gerhardt, showing a sensitive flame as affected by vibrations of different variety and intensity.

1. Gas flame. The flame is most sensitive just below the flaring point.
2. The same on a rotating mirror.

3, 4, and 5. The flame while the vowel U is loudly pronounced at 3, the mouth; 4, the trachea; 5, on the anterior chest wall.

(With right-sided pleural effusion would get 2 on the right, and 5 on the left side.)

6. Vibrations over a femoral aneurism.
7. The tympanitic tracheal tone, with a half-closed glottis.
8. The tympanitic tracheal tone, with open glottis (rhythmic vibrations).
9. The resonant non-tympanitic note of the chest wall (unrhythmic vibrations).

1. Functionally efficient vocal cords.
2. A sufficiently resonant voice: the resonator (thorax) must bear definite suitable proportions to those of the vocal cords. The existence of such a favorable relationship in men, and its usual absence in women, explains the greater intensity of fremitus in the former. The great indi-

vidual variations met with in apparently similar types of individuals have a like explanation.

3. Open, patulous bronchi.

4. Comparatively free transmission of vibrations from the lung to the chest wall; *e.g.*, the interposition of liquid, as in the case of a pleural effusion, diminishes or obliterates fremitus, unless the underlying lung is solidified, in which case fremitus remains present.

Fremitus is Normally Intense.—(*a*) On the *right side*, especially over the upper lobe, because the trachea lies in immediate contact with the apex of the lung, whereas on the left side it is separated by a distance of 3 cm. owing to the interposition of the aorta, internal carotid artery, esophagus, lymphatic and areolar tissue (Figs. 49, 52, 75, 95).

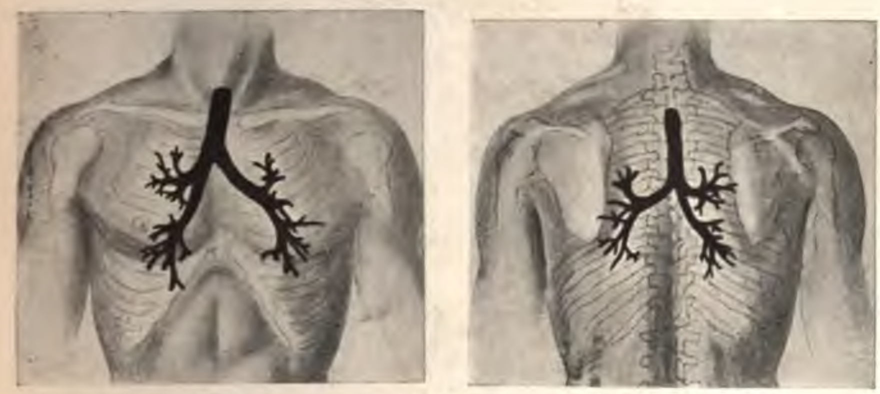

FIG. 47. FIG. 48.

FIGS. 47 AND 48.—Diagrams drawn from radiograms of the bronchi filled with a fusible alloy showing the relation of the bronchi to the chest wall. The right bronchus is larger, and a more direct continuation of the trachea—which slopes toward the right. Contrary to what is usually taught it is not more, but *less* horizontal than the left. The right bronchus gives off a branch before it enters the lung. The trachea bifurcates at Louis' angle slightly to the right of the mid-sternal line. At this point vocal fremitus and resonance are intense and the breath sounds harsh.

(*b*) *Posteriorly between the scapulæ*, because of proximity to large bronchi (Figs. 48, 91).

(*c*) In the *second right intercostal space*, owing to the nearness of the bronchial bifurcation (Figs. 47, 95, 104).

(*d*) It is more intense in the interspaces (finger tips or ulnar surface of little finger used) than over the ribs.

Fremitus is Pathologically Increased.—(*a*) Over *pulmonary consolidation*, because of better conduction of the vibrations from the bronchi (see Chap. III).

(*b*) In cases of atelectasis, because the bronchi are nearer to the chest wall.

(*c*) Over pulmonary cavities (as a rule) because of surrounding consolidation.

Increased fremitus is generally associated with bronchial breathing and bronchophony. Of course, in a given case there may be factors working at variance with one another as regards the production of bronchial breathing, bronchophony and increased fremitus. While these three signs are of the same diagnostic importance we must determine each separately.

Fremitus is *theoretically* always diminished or absent over cavities. There is little or no fremitus over the trachea, because air does not transmit vibratory waves appreciable to the hand, and because there is no reinforcement by a resonator as in case of the thorax. But *practically* we find fremitus in the majority of cases increased over cavities on account of (1) an overlying area of consolidated lung, (2) transmission by well-organized fibroid walls, (3) general sense of increased fremitus

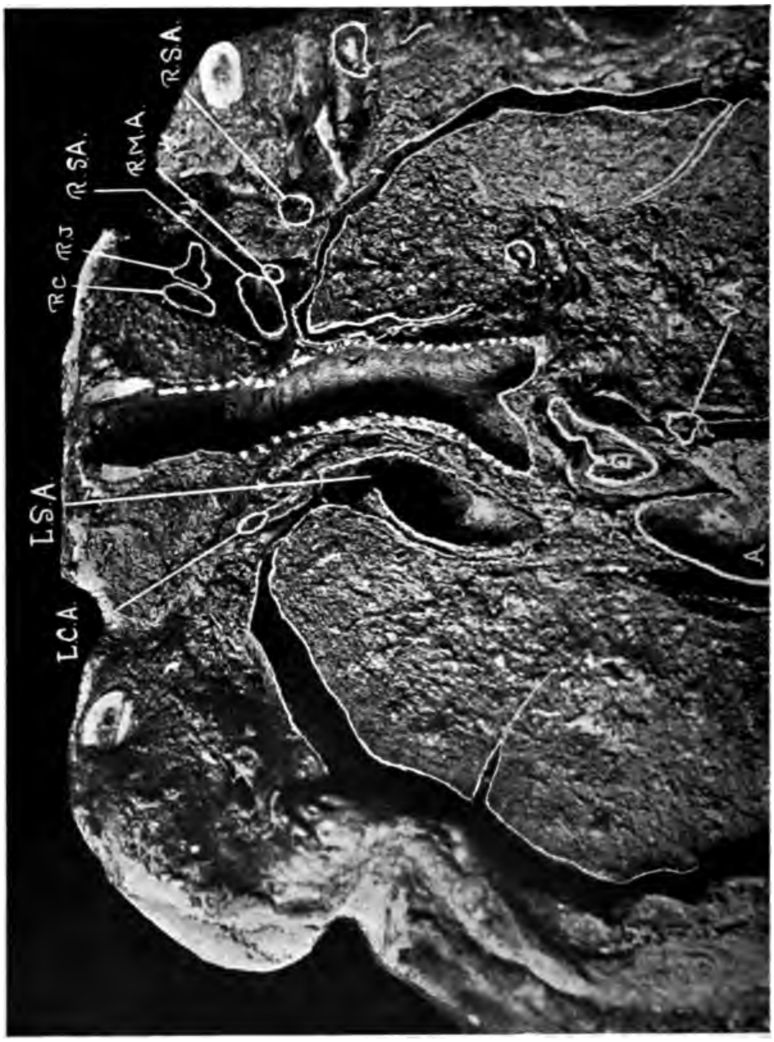

FIG. 49.—Section of the thorax, in the axis of the trachea, *viewed from behind*, showing the direct contact of the right upper lobe with the trachea and the relatively wide separation of these structures on the left side. *These anatomic differences are responsible for the more intense vocal fremitus and resonance over the right upper lobe.* R.C., right carotid artery; *R.J.*, right jugular vein; *R.S.A.*, right subclavian artery; *R.M.A.*, right mammary artery; *L.S.A.*, left subclavian artery; *L.C.A.*, left carotid artery; *V.A.*, vena azygos; *E.*, esophagus; *A.*, aorta. (Compare Figs. 52, 75.)

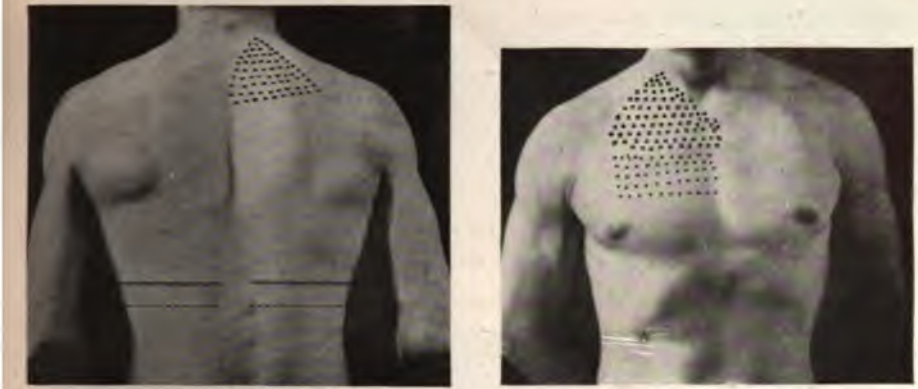

FIG. 50. FIG. 51.

FIGS. 50 AND 51.—The dots indicate the areas of the chest over which vocal fremitus and vocal resonance are normally most intense. The solid lines indicate the lower margins of the areas over which these phenomena are obtainable (lower pulmonary border). The dotted lines indicate the lower margin during forced inspiration, the lungs having expanded downward, filling the complemental space. Downward movement of the percussion resonance from the solid to the dotted lines during inspiration also indicates pulmonary expansibility.

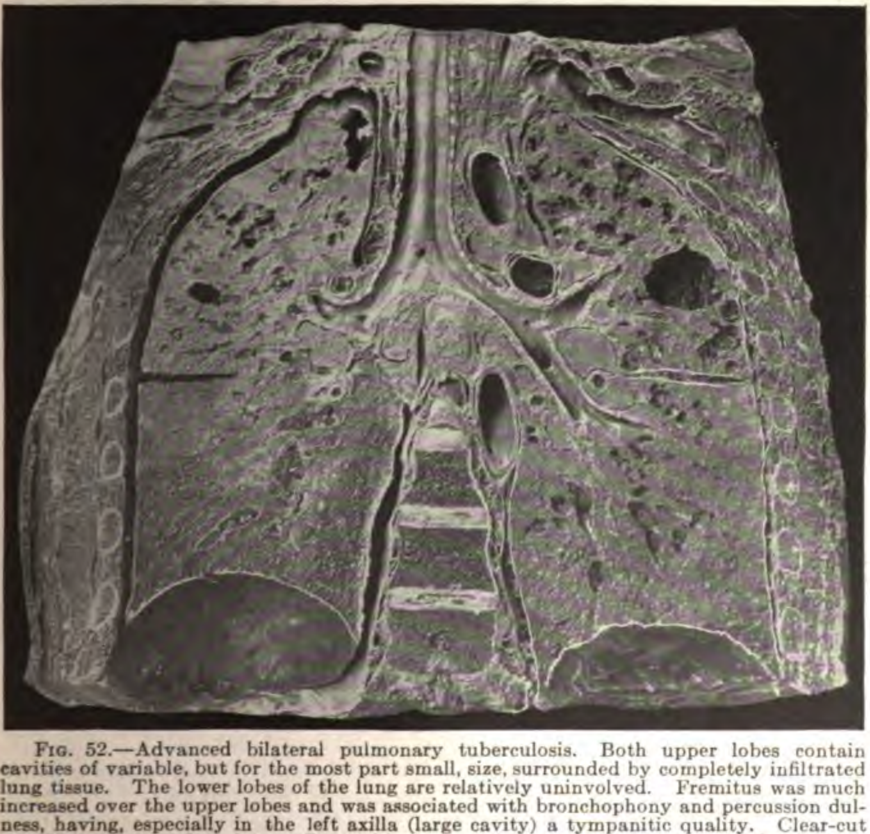

FIG. 52.—Advanced bilateral pulmonary tuberculosis. Both upper lobes contain cavities of variable, but for the most part small, size, surrounded by completely infiltrated lung tissue. The lower lobes of the lung are relatively uninvolved. Fremitus was much increased over the upper lobes and was associated with bronchophony and percussion dulness, having, especially in the left axilla (large cavity) a tympanitic quality. Clear-cut metallic râles were heard, as well as whispered pectoriloquy. The lower lobes yielded an impaired note with diffuse crackling râles. Expansion over the upper chest was almost

from surrounding consolidated lung, in other words, failure to localize the fremitus directly over the cavity.

Fremitus is Normally Feeble.—(*a*) As the result of overlying fat or mammary glands (vibratory reflection).

(*b*) In women and children because the chest, the resonator, is not in as favorable a relation with vocal cords as in men and because the voice being higher in pitch the vibrations are more rapid and hence less readily felt.

Fremitus is Pathologically Decreased.—(*a*) In obstruction of a bronchus—asthma, compression, occlusion. (In these cases it may return after coughing.)

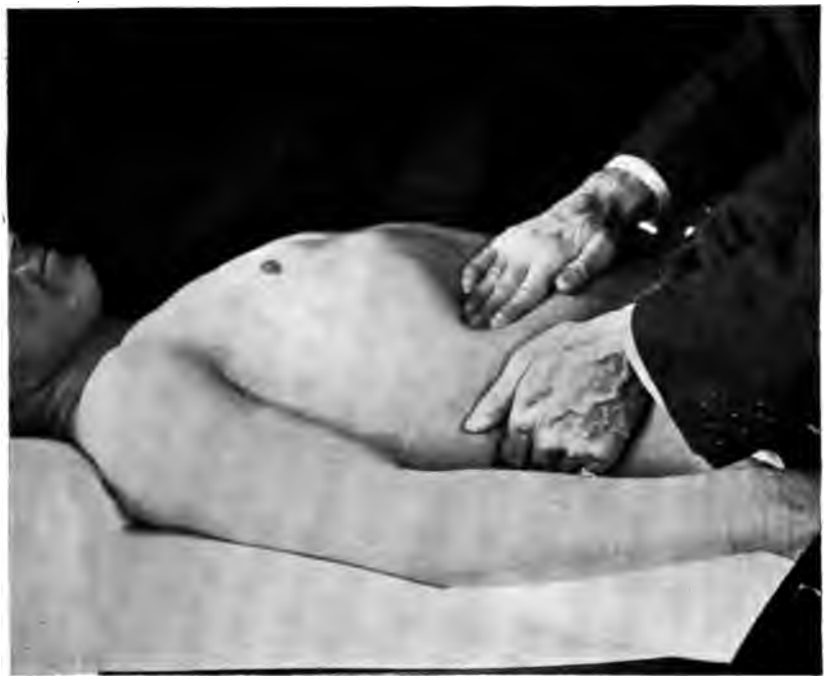

Fig. 53.—Palpation furnishes the most satisfactory method of examining the liver. The patient lies on his back with the knees slightly flexed, and is told to breathe slowly and deeply with the mouth open. If the liver is enlarged, or displaced downward, its margin can be felt to push against the right hand during the descent of the diaphragm. The examination is rendered more satisfactory if the examiner's left hand presses upward on the posterior costal margin. Splenic enlargement may be determined in a similar manner.

(*b*) From increased reflection or diffusion (pleural thickening, effusion, or pneumothorax).

(*c*) Over cavities, not surrounded by consolidation.

(*d*) In cases of dysphonia—weakness or paralysis of the vocal cords.

Exceptionally *fremitus may be present over pleural effusions;* this is due to the factors which are discussed on pages 61, 62.

The most intense fremitus is encountered in robust, deep-chested men. The lower the pitch of the voice, the slower the sound vibrations and the greater their amplitude, and hence, provided the thickness of the chest wall remains constant, the more marked the fremitus. This is especially the case on the right side, for the reasons already mentioned.

Tactile vibrations may also be produced by:

(*a*) Flowing liquids—thrill.

(*b*) Movements of fluid—fluctuation, succussion.

(*c*) Mechanical friction—friction fremitus, pleural or pericardial fremitus.

The costal and visceral pleuræ during respiration glide with smooth, mirror-like surfaces over each other. The greatest degree of excursion is vertical (3 cm.). They also move horizontally, their motion being downward and forward during inspiration (see Figs. 9, 10, 11). In health no palpable thrill or audible sound is produced. But the pleural surfaces roughened by disease may produce palpable vibrations as well as sounds (friction fremitus and friction sounds), both being most marked in the mid- and lower-axillary regions.

CHAPTER III

ACOUSTICS IN PHYSICAL DIAGNOSIS

For the ready comprehension of physical diagnosis a superficial knowledge of acoustics is necessary. The phenomena of percussion, auscultation and to a considerable extent palpation, cannot otherwise be grasped or properly interpreted. We have therefore, been led to briefly review some of the more important laws of sound. Physical diagnosis is for the most part based upon a foundation of acoustics. Our interpretation of the signs elicited from both healthy and diseased tissues is but a manifestation of the fact that physical alterations of the tissues cause corresponding changes in the vibrations which they are capable of assuming or transmitting. The fact that all the phenomena met with cannot as yet be satisfactorily explained is due to the limitations of our knowledge and is not to be attributed to any mysterious manifestations. Acoustics has not developed into such a lofty, rich, well-rounded form as has her sister science, Optics.

Rhythmic Vibrations.—When the equilibrium of an elastic body is momentarily displaced, it vibrates back and forth until its equilibrium is regained. Thus a bass viol string when on a stretch, if plucked, can be seen to vibrate. The motion imparted to a localized area is gradually transmitted throughout its whole length. The rate of the vibration is increased as the string is rendered more taut, until separate vibrations can no longer be seen. At the rate of 16 per second the lowest audible tone is produced.[1] For the human ear the highest perceptible note corresponds to 36,000 vibrations per second. The vibrations are perceptible to touch (vocal fremitus) as well as hearing, but touch perceives the slow, and hearing the rapid, vibrations more readily. The musical range of vibrations lies between 40 and 4000 per second. The greater the tension, the more rapid the vibrations, and the higher the pitch. When an elastic tissue (*e.g.* violin string), vibrates rhythmically and with sufficient rapidity a tone is produced.

But a string vibrates both as a whole, and simultaneously in its aliquot parts, at its nodal points, each of which represents an octave of the fundamental note (the note of string vibrating as a whole). These partial vibrations produce additional tones which are known as *overtones*. Overtones bear a simple relation to the fundamental note: 2, 3, 4, 5, 7, etc. The first six overtones are harmonious, above this they are generally not so. The combination of the fundamental note plus the overtones produces what is known as klang (timbre). Klang, then (the *musical quality*) results from *rhythmic vibrations* such as occur when the fundamental note vibrates together with harmonious overtones.

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[1] Not only the rate and amplitude of the vibrations, however, determines their audibility, their duration is perhaps of even greater importance. Recent investigations (Gianfranceschi) have shown that vibrations must last one-fortieth of a second to be audible, and that duration is a much more constant factor in perceptibility than rate

On different, though accurately attuned instruments—flute, violin, clarionet—the same fundamental note may be sounded, yet the individuality of each instrument remains distinct; we can distinguish one from the other. Now this individual difference—timbre, klang, quality—is dependent upon the character of the overtones. The individual quality of different voices is dependent upon similar factors.

Unrhythmic Vibrations.—Theoretically if a string could be struck at a minute point, by a hard hammer, in 0 time, the vibrations would tend to remain localized, and those points of the string which had not been directly struck would begin to vibrate slowly and unrhythmically. As a result the overtones and especially the higher overtones—the unharmonious ones—would disproportionately increase in strength and an unpleasant metallic note result (Helmholtz). In the case of the piano this is obviated by having the strings struck by soft broad hammers, which remain in contact long enough to ensure a continuous vibration of the whole string, and are so placed as to dampen the unharmonious overtones by eliminating their nodal points. If in any sound-producing body the elastic equilibrium be briefly and locally disturbed, unrhythmic vibrations result. *Very unrhythmic vibrations* allow the distant overtones to preponderate, and *a metallic quality* is produced. This occurs regardless as to whether we are practicing auscultation or percussion, and regardless as to whether these higher overtones are heard together with the fundamental (amphoric breathing) or separately. *All the metallic sound phenomena, of auscultation and percussion are thus produced* (metallic ring, bell tympany, amphoric breathing, cracked-pot sound) (see Fig. 55).

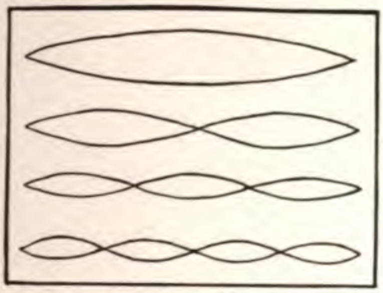


FIG. 54.—Diagrammatic illustrations of a vibrating string. First, as a whole; second, with one nodal point; third, with two nodal points; fourth, with three nodal points.

Vibrations in Tense Membranes.—Tense membranes such as a kettle drum or the distended stomach, tend to vibrate with very diverse and variable nodal points, and hence the relationship between the fundamental note and the overtones is a very variable one. This may be illustrated by throwing stones into a pool of water. Each stone will produce its own circle (vibrations) and these circles will mingle and interweave without losing the original identity.

Sympathetic Vibrations.—Vibrations may be set up in neighboring tissues not only by the direct conduction of the sound-producing impact, but also by what is known as sympathetic vibration. It has been stated that overtones accompany the fundamental note, but it is also true that if an overtone be produced the fundamental note which corresponds to it will begin to vibrate. Thus if a certain note on a piano be struck and suddenly damped, certain other strings can be heard to vibrate. If iodide of nitrogen be painted upon the "G" string of a bass viol and allowed to dry, a violent detonation will occur if a similarly pitched string of another instrument in the neighborhood is set in vibration; while vibrations of the "E" string are without effect. It is also a well-known fact that the vocal or instrumental production of certain musical notes may, to the chagrin of the musician, crack glass vases on the nearby mantelpiece. How important a rôle sympathetic vibrations play in physical diagnosis cannot be stated, but that they have some bearing cannot be questioned.

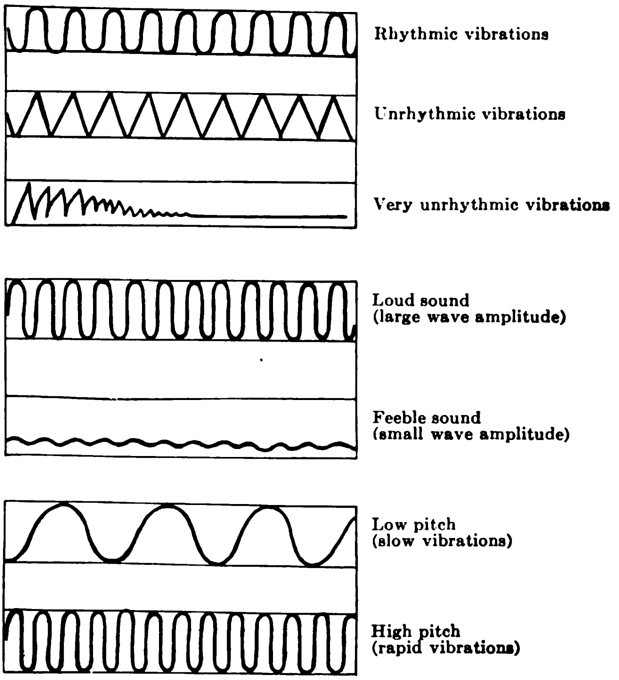
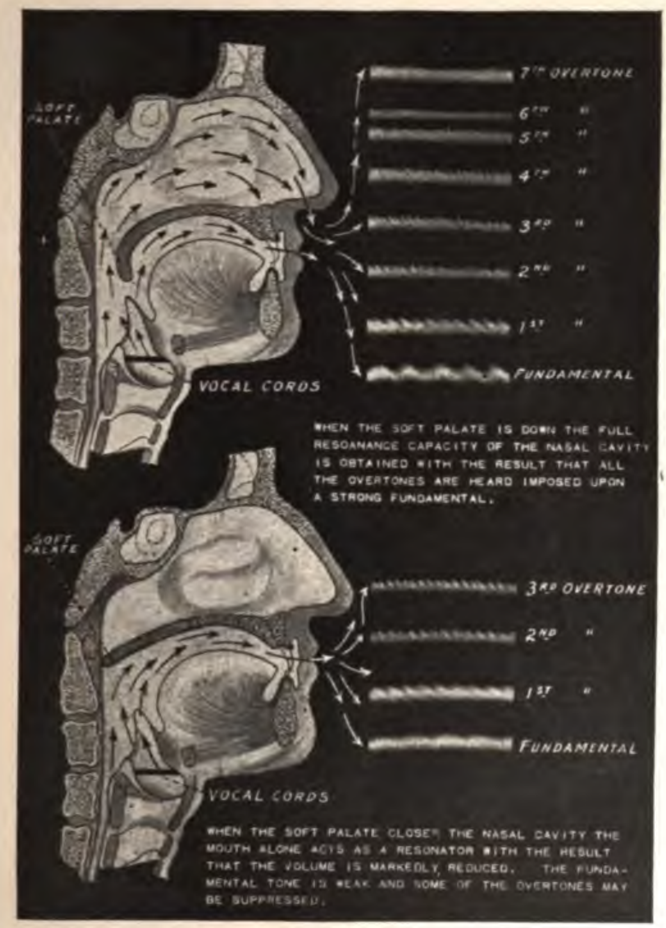

Rhythmic vibrations
Unrhythmic vibrations
Very unrhythmic vibrations
Loud sound (large wave amplitude)
Feeble sound (small wave amplitude)
Low pitch (slow vibrations)
High pitch (rapid vibrations)

FIG. 55.—Diagram illustrating the difference in the sounds produced by vibrations of different kinds.

Interference Waves.—When two waves travel through a vibrating string in opposite directions, they tend to nullify each other if their nodal points be similar. This phenomenon has been used to explain some physical signs.

Loaded Strings.—If a piece of wax be attached to one of two tuning forks, or to musical strings of a similar pitch, the vibrations of the structure thus treated become slower than those of its fellow and the note which it gives forth, correspondingly lower in pitch. A bottle filled with soapsuds gives forth a much lower note when percussed, than a similar body when empty. Here the suds act as a "load" and retard the vibra-ions. In lungs under normal tension the *pulmonary septa act as* *nd tend to localize and delay the vibrations.* The relaxed lung in he septa are no longer under tension yields a tympanitic note o igher pitch than the normal organ, and vibrates as a whole; not where it is percussed, as does the normal lung.



WHEN THE SOFT PALATE IS DOWN THE FULL RESOANANCE CAPACITY OF THE NASAL CAVITY IS OBTAINED WITH THE RESULT THAT ALL THE OVERTONES ARE HEARD IMPOSED UPON A STRONG FUNDAMENTAL.

WHEN THE SOFT PALATE CLOSES THE NASAL CAVITY THE MOUTH ALONE ACTS AS A RESONATOR WITH THE RESULT THAT THE VOLUME IS MARKEDLY REDUCED. THE FUNDAMENTAL TONE IS WEAK AND SOME OF THE OVERTONES MAY BE SUPPRESSED.

FIG. 56.—The production of overtones. Opposite the drawings are reproduc ctual photographs obtained by means of the improved Koenig manometric flan tus) the vibrations of the fundamental note and the overtones. (*From the* *merican.*)

Resonators.—Sound waves are transmitted readily from s lid bodies, as from a tuning fork to the skull, but not so readil ibrating bodies to the air—a violin string vibrating between the roduces little sound. To *increase the volume of sound*, therefo sort to the use of sound boxes or resonators, as when a tunin sts on a table. The resonators used for musical instruments sume all varieties of simple and compound vibrations which parted to them, that is they vibrate in unison with the source of the sound (sympathetic vibration) and thus amplify the waves. Indeed, it is in part due to certain vibratory habits which they have acquired that old violins owe their mellow richness of tone.[1]

Resonators are also used to *analyze sounds*—to resolve a complex sound into its component elements to a degree which is not otherwise possible. Such resonators are hollow bodies, with a smooth lining, stiff walls and two openings. The most powerful types are spherical in shape, but many forms are used, as in case of the violin. For the purpose of analyzing the sounds met with in physical diagnosis, simple telescopic tubes 2 meters in length have proved sufficient. Resonators are characterized by the fact that when a sound is produced they cause the fundamental note to predominate, while the overtones remain weak and unharmonic.

The large end of the resonator is brought into proximity with the sound to be analyzed, the small end is connected with the ear by means of rubber tubing. By varying the length of the tube to the point which produces the loudest sound for a given note, the wave length of that note can be computed and its position in the musical scale established.

In the human body the thorax, a distended stomach, a pneumothorax, etc., may act as resonators. The more elastic the material of which the resonator is composed, the longer its vibration. Thus a bell sounds longer than a glass, and a glass longer than a wooden bowl. A resonator 15 to 30 cm. in length is sufficient to amplify the sound of pure bronchial breathing (little "D" to "E" on the musical scale), but for normal vesicular breathing a length of from 1 to 2 meters ("A" to "F") is required. The latter is, therefore, from two to three octaves lower in pitch than the former.[2]

While inferior to the resonator in point of accuracy, very complex sounds can be resolved by the human ear into their component elements. The expert leader can volitionally single out the notes of any individual instrument of his orchestra, and it is the unconscious analysis of sound—the separation of the fundamental note from the overtones—which enables us to distinguish individual voices, and to recognize tympany, resonance, metallic sounds, etc., in percussion and auscultation. With percussion of equal force, the high notes are more acutely perceived by the human ear, than the low ones, which are apt to be overshadowed by their overtones. It is evident, therefore, that the individual who is the possessor of a good musical ear will much more readily acquire efficiency in auscultation and percussion, and will derive much more information from the employment of these methods, than he who is more or less tone-deaf.

THE QUALITIES OF SOUND

The qualities of sound are as difficult to describe as are colors to the blind, hence we are forced to use similes and comparisons. Sounds are classified according to their component qualities, which are ***intensity***, *duration*, *quality* and *pitch*.

---

[1] Wooden resonators, especially those made of spruce (which is made up of very long, straight, regular fibers, and stretched taut like a string) are chosen for most musical instruments, since their vibrations result in much richer, mellower tones than do those of other wood or metal.

[2] MUELLER, F.: *Zeitschr. f. Aertzliche Fortbildung*, ix, 1912, No. 14.

Intensity.—This depends upon the amplitude of the sound waves; not only on the force of the blow, but also on the number of air columns set in vibration. Hence the importance of percussion of equal force (Fig. 55).

Duration.—The more air in a vibrating column the longer the duration of the sound. The "fullness" and the "leerness" of Skoda, terms which are still occasionally employed in German literature, although compound perceptions, depend mainly upon the duration of the vibrations. *Resonant and tympanitic notes are long; dull and flat notes are short.*

Quality.—By this term we mean that a sound either is or is not musical. If it has a musical quality, we say that the sound possesses timbre or klang, attributes which depend upon the number and quality of the overtones. It must, of course, be remembered that in physical diagnosis we shall find no pure musical notes, but only varying degrees of tone dulling. The difference between resonance and tympany, between a wooden bowl and a glass, is due to the fact that the last named in each instance possesses many and harmonious overtones (Fig. 56).

Pitch.—Pitch, as was first notably emphasized by Austin Flint, is from a medical standpoint probably the *most important element in the analysis of sound.* It depends on the rate of the vibrations—the more rapid the rate, the higher the pitch. The longer the air column set in motion, the slower the vibration and the lower the pitch. Long organ pipes or long instrumental strings of equal caliber give forth the lowest sounds. The adult chest yields a lower note than that of the infant. *Increase in pitch, loss of resonance, and shortness of duration go hand in hand* (pulmonary consolidation). Auenbrugger wrote: "Ubi sonus est altior, ibi est morbus." Our recognition of the degree of pulmonary consolidation depends largely upon the high-pitched note which such tissue yields when percussed.

All sounds possess the qualities of intensity (loudness) and duration, some in addition the qualities of pitch and tone (klang).

In medical parlance we speak of "tones" and apply the term to more or less distinct resonances, but it must be remembered that none of them are tones as defined by physics, nor in the musical sense. In the latter a tone is a sound of definite pitch which cannot be further resolved into simpler sounds. "No pure tone can have timbre (klang)." Percussion sounds are always more or less muffled, impure and dull. In a strict sense there is no sharp distinction between a noise and a tone. Each can be resolved by the ear into simpler tones (Fig. 55).

Extremes are easily recognized, but there are many gradations which cannot be definitely analyzed. Even the typical tympany of an air-distended abdomen is far from having the rhythmic relations between the fundamental note and the overtones which is met with in pure musical tones. Again tympanitic, and non-tympanitic notes often merge so gradually into each other that an absolute differentiation is impossible. In the case of a tympanitic sound, the fundamental note is readily
an sing it, but with a non-tympanitic note this is not the case.
ndary between the two lies at the point at which the ear fails to
pitch, on account of the interference of the "overtones," and this
question of the individual ear.
we strike a key on the piano a tone results, but if we strike a
keys, musical notes blend to form a noise. Certain of the modern
re noises to the uneducated ear.[1]

L: *Deut. Arch. f. kl. Med.*, vol. lxxxviii, p. 598.

THE ORIGIN OF SOUNDS HEARD OVER THE CHEST

The act of speaking or breathing produces sounds in the upper respiratory tract—larynx, vocal cords, glottis, mouth, nares—which are conducted downward into the chest mainly by the air columns in the lumen of the bronchi. The moving column of air sets the pulmonary tissues into vibration, and vibrations of certain kinds produce audible sounds.

These sounds are conducted through the overlying tissues to the ear of the examiner, and the character of the sound we hear depends upon the character of the tissues in their effect upon the following acoustic factors: (1) *diffusion,* (2) absorption, (3) *reflection,* (4) resonance, and perhaps (5) interference.

The Diffusion of Sound.—By this is meant a loss of intensity due to conduction over a wide area—a dilution of energy. In spreading from a sphere of a given size to one double its size, the intensity on the surface of the second sphere, the vibratory energy per unit area, will be halved. The function of the stethoscope or of a megaphone is to diminish diffusion (lateral radiation). *Diffusion appears to be the most important factor in reducing the intensity of sound heard over the chest* (Montgomery).

The Absorption of Sound.—By this term we refer to the annihilation of sound as such, and its conversion into forms of energy such as heat, of which the ear takes no cognizance. It plays but a small rôle if sounds travel such short distances as is the case in the chest. Its effects are separate and distinct from those of diffusion.

The Reflection of Sound.—Sound waves may be reflected backward toward the direction from which they come. This is especially apt to occur *when vibrations pass through tissues of different densities,* as for instance when air-borne vibrations pass from the lumen of a bronchus to its walls or from the alveoli to fluid or to the chest wall. The degree of loss of sound depends "upon the differences in density and elasticity of the two media, irrespective of whether the sound is passing from the heavier to the lighter, or from the lighter to the heavier medium, provided the sound passes from one medium to another at the normal incidence, and provided that plane surfaces come into contact or separate two media, and provided that the two media are homogeneous and infinite in extent" (Montgomery).

Resonance.—By resonance we mean *tone reinforcement,* due to the fact that amplifying vibrations are set up in a second medium so that the two media vibrate in unison and a louder sound results.

Although playing a less important rôle in modifying the acoustic phenomena in the chest than do diffusion and reflection, yet resonance at times is an important factor. It is to be noted that whereas diffusion, absorption and reflection modify all sounds in like degree providing that intensity is constant, resonance is selective, and amplifies sounds of different pitch (vibratory rate) in very different degree. "Conditions favor-

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FIG. 57.—Diagrammatic representation of how reflection and diffusion, the most important factors that diminish sounds on their way through the chest, may operate in some pulmonary and pleural conditions. (*After Montgomery and Eckhardt,* "Pulmonary Acoustic Phenomena," *10th Report, Henry Phipps Institute, Phila.,* 1915.)

The continuous lines indicate surfaces of special reflection; the broken lines indicate the surfaces of no special reflection except those common to all the conditions, namely, the outer chest surface and the internal bronchial surface. Fluid-tissue junctions, and the junctions between relaxed lungs and air, are not specially indicated, because the changes in density at these junctions are not specially great.

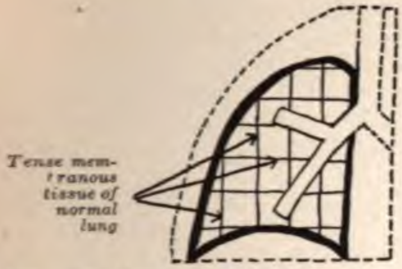

FIG. *A.—Normal lung.* Reflection occurs at lung-chest junction, at junction of tense membranous tissues of lung with air, and at surface of contact between bronchi and surrounding air in the vesicles (a somewhat questionable factor). Diffusion much as in Fig. *B.* Vocal fremitus and resonance normal. *Breath sounds "vesicular."*

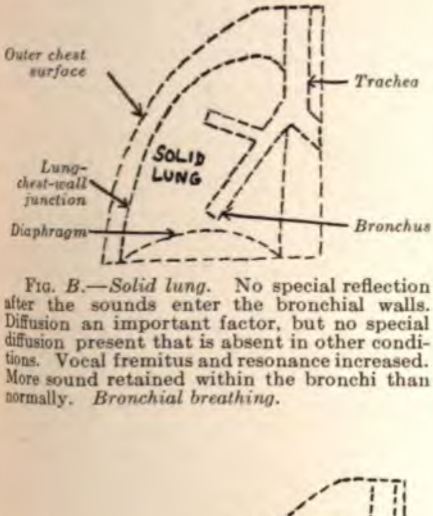

FIG. *B.—Solid lung.* No special reflection after the sounds enter the bronchial walls. Diffusion an important factor, but no special diffusion present that is absent in other conditions. Vocal fremitus and resonance increased. More sound retained within the bronchi than normally. *Bronchial breathing.*

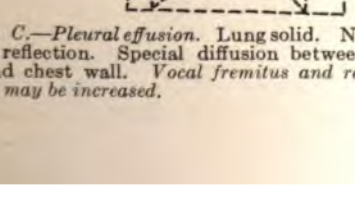

FIG. *C.—Pleural effusion.* Lung solid. No special reflection. Special diffusion between lung and chest wall. *Vocal fremitus and resonance may be increased.*

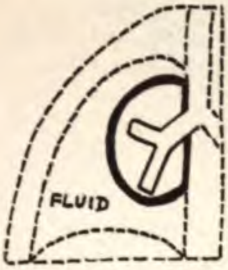

FIG. *D.—Pleural effusion.* Lung air-bearing, but membranous tissue relaxed. Special reflection at lung-fluid junction, and, to some extent, at surface of contact between bronchi and surrounding intravesicular air. Special diffusion between lung and chest wall. Vocal fremitus and resonance diminished. *Breath sounds feeble or absent.*

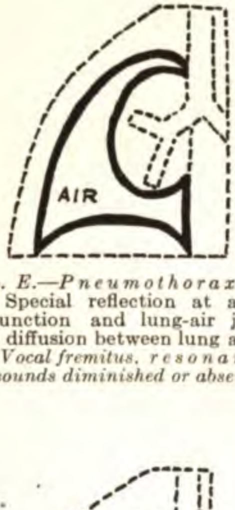

FIG. *E.—Pneumothorax.* Lung solid. Special reflection at air-chest-wall junction and lung-air junction. Special diffusion between lung and chest wall. *Vocal fremitus, resonance and breath sounds diminished or absent.*

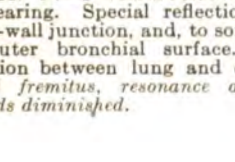

FIG. *F.—Pneumothorax.* Lung air-bearing. Special reflection at air-chest-wall junction, and, to some extent, at outer bronchial surface. Special diffusion between lung and chest wall. *Vocal fremitus, resonance and breath sounds diminished.*

able to the resonance of sounds of one pitch may actually diminish sounds of another pitch. In addition sounds from different sources may come together, and thus sounds will be superimposed upon one another, and a condition known as *interference* may result," causing blurring of sound. As an example of this we have the fact that vocal resonance may be diminished, and yet the whispered voice sounds present, over a pleural effusion.

The importance of resonance or vibratory amplification in regard to vocal fremitus has been considered in the preceding chapter.

"**Normal Lung.**—The diminution of sounds on their way through the normal chest is due chiefly to the factors of *diffusion* and *reflection.* The latter acts particularly: (1) As the sound within the air in the bronchi comes into contact with the bronchial walls, especially of the larger bronchi; (2) as the sound passes from bronchial wall to the air in the surrounding vesicles; (3) as the sound comes into contact with the tense pulmonary membranes; and (4), as the sound passes from the air in the lung to the chest wall (Fig. 57, *A*).

"**Consolidation.**—Apart from the marked loss in intensity of the sounds as they pass from within the bronchi to the bronchial walls, the chief loss of sound in consolidation as it passes to the periphery of the chest is due to diffusion. It is not due to absorption. One can readily see why the solid lung ordinarily gives louder sounds than the normal lung (Fig. 57, *B*).

"**Pleural Effusion.**—In cases with diminished or absent vocal resonance the chief source of sound loss after the vibrations have entered the fluid is due to diffusion, and not to absorption or reflection. Diminution of sound is impeded, and there *may be an actual increase in vocal resonance* when the lung, instead of being collapsed and air-bearing, is actually solid, because there is comparatively little sound lost at the lung-fluid junction when the lung is solid, just the reverse of what occurs when the lung contains air (Figs. 57, *C* and *D*).

"**Pneumothorax.**—The conditions here are roughly parallel to those encountered in pleural effusion, provided the lung is air-bearing and is equidistant from the chest wall in each case, except that with pneumothorax a marked loss in sound intensity, instead of occurring at the lung-fluid junction, is met with at the air-chest-wall junction. If the lung in the case of pneumothorax is solid, a very serious loss in sound intensity will take place at the lung-air junction. If an open fistula between a bronchus and the pleural cavity exists, the vocal resonance will be increased" (Montgomery) (Figs. 57, *E* and *F*).

The inspiratory vesicular sound *arises within the lungs at the point at which the air passes from the smallest bronchioles into the alveoli.* It is ordinarily independent of the sounds arising in the upper respiratory tract.

The expiratory vesicular sound arises partly if not entirely *in the upper respiratory tract* (nares, mouth, glottis, larynx) (Fig. 56, 96).

The sounds which we hear in *bronchial breathing* arise mainly in the upper tract. The solidified lung, though causing no new sounds may modify those already produced above. The same facts apply to *cavernous and amphoric breathing;* the cavities which yield them modify an already existent sound by acting as resonators. New sound production due to the air entering or leaving an excavation is generally if not always a negligible factor.

The preceding facts explain why the physical signs over what appear clinically to be identical conditions at times vary diametrically. Thus *some cases of pleural effusion yield diminished, or absent, fremitus, vocal resonance, breath sounds and whispered sounds, while in other clinically identical cases these signs may not only be present but at times actually increased.* These discrepancies are mainly due to the fact that in the former instance the lung is air-bearing—merely relaxed, while in the latter the lung is solidified, either as a result of compression or consolidation. Analogous variations occurring in cases of pneumothorax are to be explained upon the same basis.

The effects of these differences in the density of the tissues upon sound reflection and diffusion are accountable for the difference in the findings.

CHAPTER IV

THE HISTORY AND THE THEORY OF PERCUSSION[1]

The theory of percussion is based upon the fact that when elastic matter which is capable of adequate vibration is struck, a sound will be produced. Thus by striking a blow on the chest wall a sound is generated which varies with the character of the tissues within the thorax. Percussion although practised from early Grecian times for the differentiation of ascites from tympanites, was not used for thoracic diagnosis until 1755. Leopold Auenbrugger (b. Gratz, 1722) was, when about thirty years of age, put in charge of a Spanish Military Hospital in Vienna, and while doing his duties there, invented the art of percussion."

The principles of Auenbrugger's discovery are mainly two: first, that *percussion sounds are simply manifestations of acoustic phenomena which should be expressed in corresponding terms;* second, that the *variations of sounds are due to a physical variation of the tissues.*

In 1826 Piorry, in Paris, erroneously tried to establish the identity of sounds as specific of certain tissues, *e.g.*, a pulmonal, cardial, osteal, or intestinal note. The subject was put upon a permanently fixed basis by Skoda of Vienna, who in 1839 wrote: "We must first determine every possible variety of percussion sound and ascertain the conditions on which each variety depends; and then endeavor to reconcile our observations with the well-ascertained laws of sound."

PERCUSSION SOUNDS

If we percuss the chest a sound will be produced, the character of which will depend upon the amount of air, fluid and elasticity in the lung. The sounds which are thus elicited are arbitrarily *classified* according to their acoustic properties as: (1) tympany, (2) hyperresonance, (3) resonance, (4) dulness, (5) flatness.

No sharp line of distinction can be drawn between these sounds. They merge gradually into each other. What for instance one examiner might consider tympany, another of equal experience might declare to be hyperresonance. As a general rule, however, distinctions sufficiently accurate for clinical purposes, which will be concurred in by other reasonably skilled examiners, can readily be established.

Tympany.—Tympany is a *musical note* in which, although rich in overtones, the fundamental note can be more or less clearly recognized. We can sing a note to correspond with its pitch. Its name is derived from the tympanum—kettle drum. It occurs:

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[1] Students and practitioners of medicine who are interested in physical diagnosis should read the translation of AUENBRUGGER's and LAENNEC's articles, which are readily available in CAMAC's "Epoch-making Contributions to Medicine, Surgery and the Allied Sciences," Philadelphia, 1909; as well as FLINT's "Auscultation and Percussion," a recent edition of which has been revised by H. C. THATCHER, M. D.

(*A*) When air in closed chambers vibrates in unison with an elastic membrane (in an air-filled stomach or intestine).

(*B*) When air columns vibrate in smooth-walled chambers which communicate with the atmosphere (open pneumothorax).

(*C*) In relaxed lung tissue (when the lungs are removed from the body, or when their tension is relaxed by a pleural effusion, or by upward displacement of the diaphragm). The lung under these conditions vibrates as a whole, not merely locally, the percussion stroke produces *rhythmic vibrations*. A non-tympanitic (normal) lung becomes tympanitic when the internal air tension is relaxed.

Percussion of a stale loaf of bread yields tympany; of a fresh loaf, resonance. An air-inflated bladder under moderate tension yields a long tympanitic sound; a tensely inflated bladder gives forth a shorter, higher pitched, more metallic, less tympanitic note.

"The tympanitic character of the note in the first instance (*A*) is due to the fact that the comparatively relaxed and yielding wall permits a sufficiently long duration of the contact between the wall and the percussing instrument, for the movement to be transmitted to the whole bladder, so that it vibrates primarily as a whole, as does a string struck with a cushioned hammer. If, on the contrary, the bladder be markedly distended, contact is so short as to give rise to a circumscribed undulation of the surface, before the movement has reached the more distant parts of the bladder. In this case so many different portions of the bladder vibrate independently that a vast number of overtones are produced and the sound is a mere noise" (Sahli) (Fig. 58).

Lungs removed from the body yield a tympanitic note, because they have lost their tonicity and diminished their bulk. Pulmonary tone is mainly due to stretched elastic tissue, also in part to activity of the bronchial muscular fibers.[1] Relaxation or loss of tone allows the lung to vibrate as a whole. It is not the air that vibrates as a whole, because ligation of the trachea under these conditions does not alter the pitch, nor yet the elastic tissue as a whole, for this is relaxed and does not vibrate at all.

If we reinflate the lung removed from the body it becomes non-tympanitic (relaxation has given place to tension) unless the lung be emphysematous and have lost its elasticity. This is because with tension present, unrhythmic vibrations are more readily produced, which permit the overtones to predominate and render the fundamental note less perceptible, and hence the pitch is less recognizable.

A lung section held free in the air gives a lower note than when laid in a plate, because the weight of the lung of itself causes an increase in tension. If a lung be cut in longitudinal sections the pitch of each will be higher, the smaller the section. If these sections are again laid in apposition a lower pitch will result than the note of any one. The rate of vibrations in each section is roughly approximate, inversely, to the square of the thickness (Zamminer and Seitz).

It is impossible either by strong or light percussion to alter the pitch of a tympanitic lung. The only difference is that of loudness, because the lung under these conditions always vibrates as a whole. *We must not imagine, therefore, that by light percussion over a relaxed lung, as in pleural effusion we are eliciting only the note of the superficial lung tissue.*

A tympanitic note is one in which unrhythmic vibrations are impossible. It is evident, therefore, that *accurate topographic percussion* (the outlining of dull areas) *is possible only in the absence of tympany.* This explains why small broncho-pneumonic areas or tuberculous consolidations are so difficult to demonstrate by percussion in emphysematous subjects in whom more or less tympany is always present. For a similar reason dull areas under the abdominal wall, etc., cannot be outlined by percussion

Tympany is normally heard on percussing the abdomen, larynx or trachea, and over the lower anterior and lateral margin of the left lung owing to the proximity of stomach and intestines.

Pathologically tympany is heard:

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[1] WEST: *Trans. Med. Chir. Soc.*, 1898, p. 273.

1. In *consolidation overlying air chambers* (bronchi, cavities)—a dull, high-pitched tympany (Fig. 61).

2. Over *cavities* (air-containing and at least the size of a walnut) as in tuberculosis or bronchiectasis; also often over a pneumothorax (Fig. 236).

3. Over *relaxed lung tissue:* in the neighborhood of pulmonary infiltrations, and above pleural or near pericardial exudates (*Skodaic tympany*) (see Fig. 107, 339). Occasionally also in incomplete pulmonary consolidation—air and fluid, *e.g.*, edema of lungs.

4. Over areas of *subcutaneous emphysema.* In such cases if this condition overlies the lungs, no satisfactory percussion or auscultation data regarding the state of the lungs can be obtained.

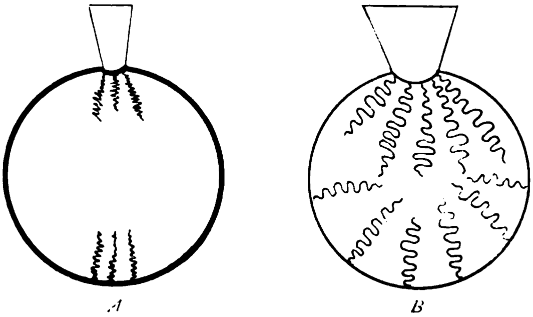

FIG. 58.—Diagram to illustrate the difference in effect of large and small pleximeters, as well as of tense and of flaccid membranes.

In *A* the membrane is stiff and tense, the pleximeter small, the percussion stroke staccato The vibrations tend to remain localized and are very unrhythmic, hence metallic sounds are produced.

In *B* the membrane is flaccid, the pleximeter broad and the percussion blow of average duration. The vibrations which result are widespread, as well as rhythmic, and hence the sound is musical (tympanitic).

The pitch of tympany varies, but as a general rule, it is higher than that of resonance. Percussion of the stomach, for instance, produces very rhythmic, regularly recurrent vibrations at a rate of 320 to 450 per second. The note is, therefore, much higher than that of the lung—108 to 54 per second (F. Mueller). In either case the pitch depends largely upon the size and tension of the organs in question.

Resonance is a *long, clear, low-pitched non-tympanitic sound.* It is due to amplification of the sound waves, reinforcement of tone (see Resonators). The percussed lung yields the fundamental note and the thorax acts as the resonator. When the two vibrate in unison resonance is produced, mere reflection of the waves is insufficient. The tissues must possess more or less rhythmic vibrations with the reflected waves in order to produce resonance. *Pulmonary resonance* is best elicited in normal lungs in the left infraclavicular region and at the angles of the scapulæ. It has been likened to the sound of a drum covered by a blanket (Auenbrugger), and to the upper crust of a loaf of fresh bread (Flint).

Resonance yields both a longer and a louder note than dulness: R. 0.42, D. 0.28 second. (Resonance is not due to vibrations in the bronchi; these structures may be filled with gelatin and yet resonance persists. Not so the alveoli.) The note over a normally distended healthy lung, in the regions in which it is neither unduly encroached upon by organs, or overlaid by tissues, is resonance. This can be more or less fixed in the mind by frequent examinations of normal chests, with the reservation, however, that there is no absolute normal standard, but merely relatively normal variations. In disease the sound side if such there be, must always be compared with the side which is diseased.

Selling has proved by means of resonators that the pulmonary note is a very complex one, made up of many overtones of which the lower ones predominate. The range of pitch of normal resonance is from low F to high C. In healthy adults it goes down to about low A; in children to middle F, in emphysema to low F. A dull note on the contrary is higher in pitch since the deeper tones are subdued through the decreasing vibratability of the lung.

The upper range of the pulmonary percussion note represents the pleximeter, the lower range is the lungs' own note. The latter varies with the amount of air and elasticity. The fact that the note between the large right and smaller left lung is not more noticeable, is probably due to the fact that when the note is low, a considerable difference in volume must occur before an appreciable change of the whole note is produced. *When infiltration of lung tissue becomes complete we practically hear only the note of the pleximeter (finger).*

In the lungs the pulmonary septa modify the sound by limiting—localizing—the sound waves; not, however, by acting at nodal points—they are too closely spaced—but by acting as a "load" on a vibrating string (see p. 56). If the lung is under normal tension, the vibrations remain localized in the area percussed. If it is relaxed, both lung and its contained air vibrate as a whole and tympany results.

Resonance occurs only over the lungs (elastic air-containing organs). The larger the quantity of air, the more resonant the note, especially if its deep diameter lies in the vertical plane of the percussion stroke.

Hyperresonance.—By this term we understand a sound which acoustically lies between resonance and tympany, having some of the qualities of each but failing to be identical with either. It may be produced by percussing the normal chest during forced, deep, held inspiration (increased tension, and increased air), but is most characteristically heard in cases of pulmonary emphysema (increased air and diminished tension). The same concept may be expressed by "resonance with a tympanitic quality."

Dulness.—A dull note is a *short, high-pitched, non-musical sound.* Percussion resonance disappears over the lung, and dulness takes its place as the result of the following physical conditions: (1) Absence of tone-producing material (consolidation of pneumonia, tuberculosis, infarction, atelectasis). (2) Poor tone conduction. (3) Poor conduction of the percussion stroke (exudation between the lung and the pleximeter—pleural thickening, effusions, edema of the skin). Dulness is "the sound of both fluids and solids, that of the airless viscera—liver, heart, spleen" (Da Costa).

Pulmonary dulness (lack of resonance) indicates an abnormally large proportion of solids or liquids in proportion to the amount of air in the pulmonary vesicles. With the appearance of dulness there is *always an increase in the elevation of the pitch*, and an *increased sense of resistance.* Acoustically, dulness is a condition in which the fundamental note preponderates, and but few overtones are heard.

A hand placed on a vibrating glass deadens the overtones. Its musical quality disappears, we hear only the fundamental note. Air-containing organs such as the intestines are more elastic, vibrate more readily and more complexly, furnish more overtones which produce a clearer, louder, longer, higher pitched, more intense and more musical sound (tympany). Solid organs on the other hand—liver, spleen, thigh—yield but few overtones, hence the sound is shorter, weaker and more muffled. Percussion of normal lung produces a larger wave amplitude—louder sound—than that of airless structures.

The dull note obtained by percussing pulmonary consolidation is characterized by the predominance of high-pitched sounds, those of lower pitch being weak or inaudible, and since high-pitched sounds fade out more rapidly than do low-pitched ones, the sound is not only high-pitched but also short, and does not carry far. The metallic, crackling râles heard in the neighborhood of pulmonary consolidation are even higher in pitch than the bronchial breathing which accompanies them (F. Mueller). The normal lung, under normal tension (inflation), yields on percussion, a long, low-pitched, resonant, non-musical note. The pleximeter finger recognizes distinct elasticity (normal resistance). The breath sounds, except near the large bronchi or the trachea, are "vesicular"—soft, breezy and low in pitch. Expiration is much shorter than inspiration. Over the lower lobes the percussion note is longer and lower-pitched than over the upper lobes because of (1) the greater mass of lung tissue thrown into vibration; and (2) thinner overlying tissues. The liver on the right and the stomach on the left tend to add respectively a dull, and a tympanitic, quality to the lower pulmonary margin.

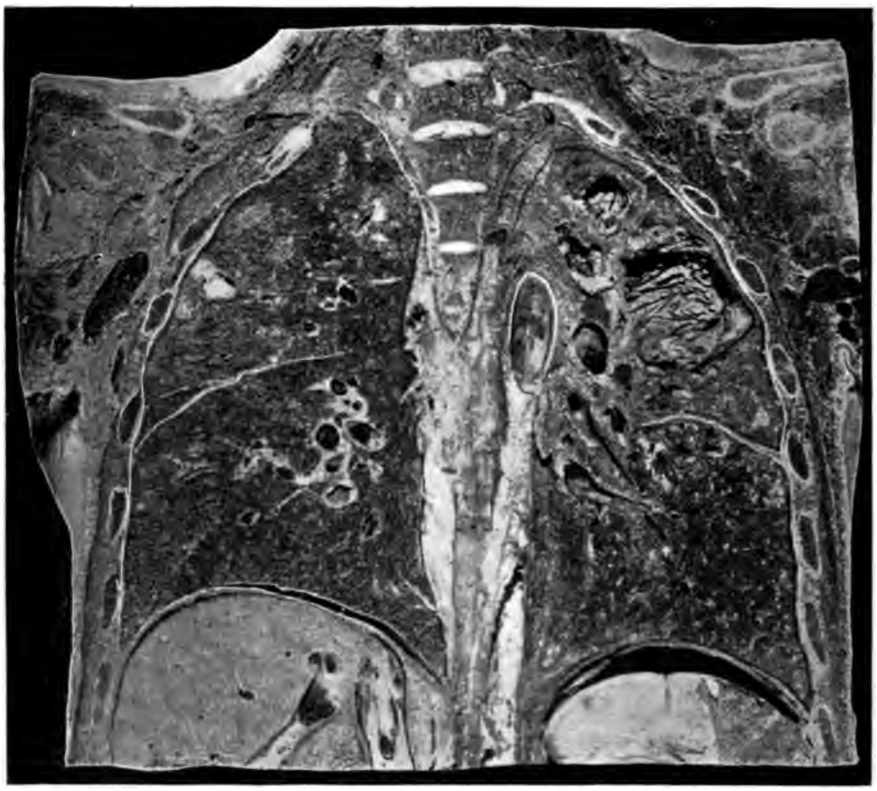

FIG. 59.—Showing different degrees of *percussion dulness in a case of pulmonary tuberculosis.* The *left upper lobe* contains several cavities filled with blood clot and surrounded by consolidated lung, yields a very dull note. The *right upper lobe* is diffusely infiltrated, yields moderate dulness. The *left lower lobe* is slightly infiltrated and yields an "impaired" note. The *right lower lobe* is relatively free from disease and yields a resonant note.

Impaired resonance or impairment is a term sometimes used to indicate slight dulness. It is somewhat shorter, high-pitched and less intense than resonance and is produced by the same causes, in lesser degree, which produce dulness—slight pulmonary infiltration, etc.

Flatness is a term which is applied to extreme or *absolute dulness.* The note of the thigh or of a fluid—pleural effusion—is flat. Some physicians prefer to use the expressions slight, marked and absolute dulness, to those just discussed. Efforts have been made to numerically classify or standardize different degrees of dulness as dulness number 1, 2, 3, 4.

Thus some examiners classify lack of resonance as 1- 2- 3- 4-line dulness, using these terms to indicate respectively impaired resonance, dulness, marked dulness and flatness. This method of recording has not been satisfactory, owing to lack of standardization. The 3-line dulness of one examiner might be called 2- or 4-line dulness by another. It has been suggested[1] that since these different degrees of dulness represent varying degrees of pitch, they might be standardized according to a musical scale, *i.e.*:

1. Normal resonance = F or F sharp below middle C.
2. 1-line dulness = A below middle C to the latter.
3. 2-line dulness = E flat to F above a middle C.
4. 3-line dulness = B flat to intermediate C.
5. 4-line dulness = E–F above intermediate C.

While such standardization would be most desirable, we cannot but question tis feasibility especially among "non-musical" examiners.

MODIFIED TYMPANY

The tympanitic or musical sound already described sometimes undergoes certain modifications which give it distinct and separate qualities, producing what are known as (1) *metallic ring;* (2) *bell tympany;* (3) *cracked pot sound.* We have already learned that if vibrations are imparted to an air-containing cavity by a steady stroke applied to a large surface, rhythmic vibrations will be set up throughout the structure. If, on the other hand, a quick, sudden, localized blow be struck, the vibrations will tend to remain localized, will be unrhythmic and will produce a harsh, metallic, unpleasant sound. This may be illustrated by striking a piano string with a knife blade.

The conditions necessary for the production of markedly unrhythmic vibrations, and hence metallic sounds (a sudden localized blow, and a quick rebound) are most nearly fulfilled in the case of a large, tense, superficial cavity. Long before an explanation of the genesis of these sounds was forthcoming, Wintrich pointed out that metallic sounds could be more easily demonstrated by quick, forcible percussion, and when a hard, artificial pleximeter was employed. To produce metallic sounds we must have a stiff walled resonator which reflects better than it transmits. If the cavity is deeply situated, too much lateral radiation of the vibrations will occur before the cavity is reached, and vibrations instead of locally, will be set up generally, throughout it, producing a tympanitic note. If the cavity is small, the fundamental vibrations will be very rapid, and the overtones still more so. It is possible that a small cavity may actually yield a metallic sound which we merely appreciate as tympany because the unharmonious overtones are above our auditory range (36,000 per second).

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[1] Wood, N. K.: "Percussion of the Lung." *Jour. Amer. Med. Assn.*, lxiii, 1914, 1378.

Metallic ring is a term which is applied to a percussion note having a metallic quality. It may be heard:

1. Over cavities not less than 4 cm. in diameter, with smooth, tense walls, especially if superficially located (Fig. 61).
2. Over a tense pneumothorax (Fig. 106).
3. Occasionally over intense apical infiltration. In this case, especially if right-sided, the trachea acts as the "large cavity with tense walls."
4. At times over a greatly distended stomach.

Metallic ring is best heard with staccato percussion and if stethoscopic auscultation be simultaneously practised.

Metallic ring occurs only over fairly large cavities because:

1. Small cavities vibrate too readily as a whole, and because their overtones are too rapid—high-pitched—to be perceptible.
2. Metallic ring is often associated with a short, high-pitched note which is closely allied to flatness.

Examples: Percussion of a thin tumbler yields tympany. Percussion of a thick bottle yields metallic ring. The fundamental note grows deeper as the size of the bottle is increased and its opening diminished. The metallic quality is especially noted if the ear be held near to the open mouth of the bottle (or of the patient, in case of a patulous cavity).

Percussion of a moderately inflated pig's bladder, the cheeks, or the stomach yields tympany; with extreme inflation the note becomes metallic.

Bell Tympany.—Bell tympany is a *clear, vibrant, metallic sound* which may be heard by ausculting over tense pneumothoraces while percussion is practised on the opposite side of the lung, using silver coins as plexor and pleximeter. A clear-cut, slightly echoing sound is thus produced which has been likened to the sound of a gold coin dropped upon a marble slab, or the sound of a distant trolley bell. Acoustically the sound owes its character to the fact that sustained high-pitched overtones vibrate together with a deep fundamental note, and fade out slowly. Coins are used for percussion since being small, hard and metallic they tend to impart unrhythmic vibrations to the tissues beneath.

Cracked-pot Sound.—This is a *metallic note, followed by a stenotic murmur,* caused by the rapid expulsion of air, through a slit-like opening, as the result of the percussion blow.

"Though obviously related to metallic resonance, the nature and causation of cracked-pot sound are essentially different. The expulsion of air through a narrow opening plays a part in causing it. Apparently the edges of the opening are set in vibration, and an opportunity is thus given for the production of high overtones by interference" (Sahli).

Cracked-pot sound (bruit de pot fêlé) then is heard: (1) when air is forced through a stenotic opening as in the case of a superficial cavity communicating with a bronchus (vibrations of the tissues at the mouth of the cavity); (2) over the chest of a crying child (glottic vibrations); (3) occasionally over relaxed and infiltrated lung tissue. It is most marked if the examiner's ear be close to the patient's open mouth and during expiration. It is best heard over a superficial cavity near the pulmonary apex, with tense but resilient walls and a stenotic outlet. The sound may be imitated by striking the loosely clasped hollowed palms of the hands together against the knee, or by suddenly percussing a perforated rubber ball. *All metallic sounds are best elicited if forcible staccato percussion is employed.*

Thus we find that: Rhythmic vibrations produce tympanitic sounds.
Unrhythmic vibrations produce non-tympanitic sounds.
Markedly unrhythmic vibrations produce metallic sounds.

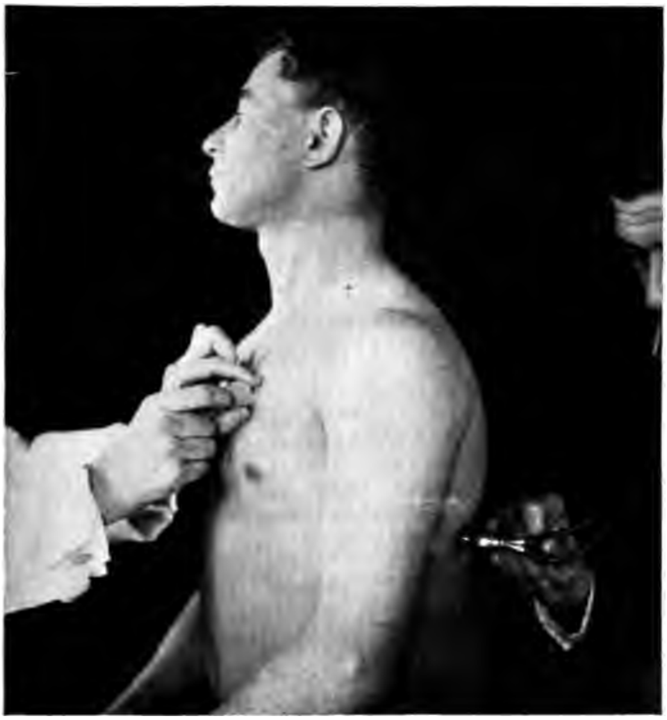

FIG. 60.—The coin test.

SUMMARY.—1. **Resonance** is a *long, low-pitched, non-musical note,* heard on percussing normal lung, due to unrhythmic, more or less localized vibrations. It may be imitated by percussing a loaf of fresh bread. It is associated with a sense of resiliency.

2. **Tympany** is a long *musical note* of variable pitch, produced by percussing elastic, air-containing viscera. It is due to widespread rhythmic vibrations and may be heard over the stomach, intestines, over relaxed lung tissue, and over many cases of pulmonary cavitation and pneumothorax. It may be imitated by percussing a loaf of stale bread or towel folded many times upon itself, or the inflated cheeks.

3. **Dulness** is a *short, non-musical,* invariably *high-pitched* sound, heard when percussing structures containing little or no air. It is due

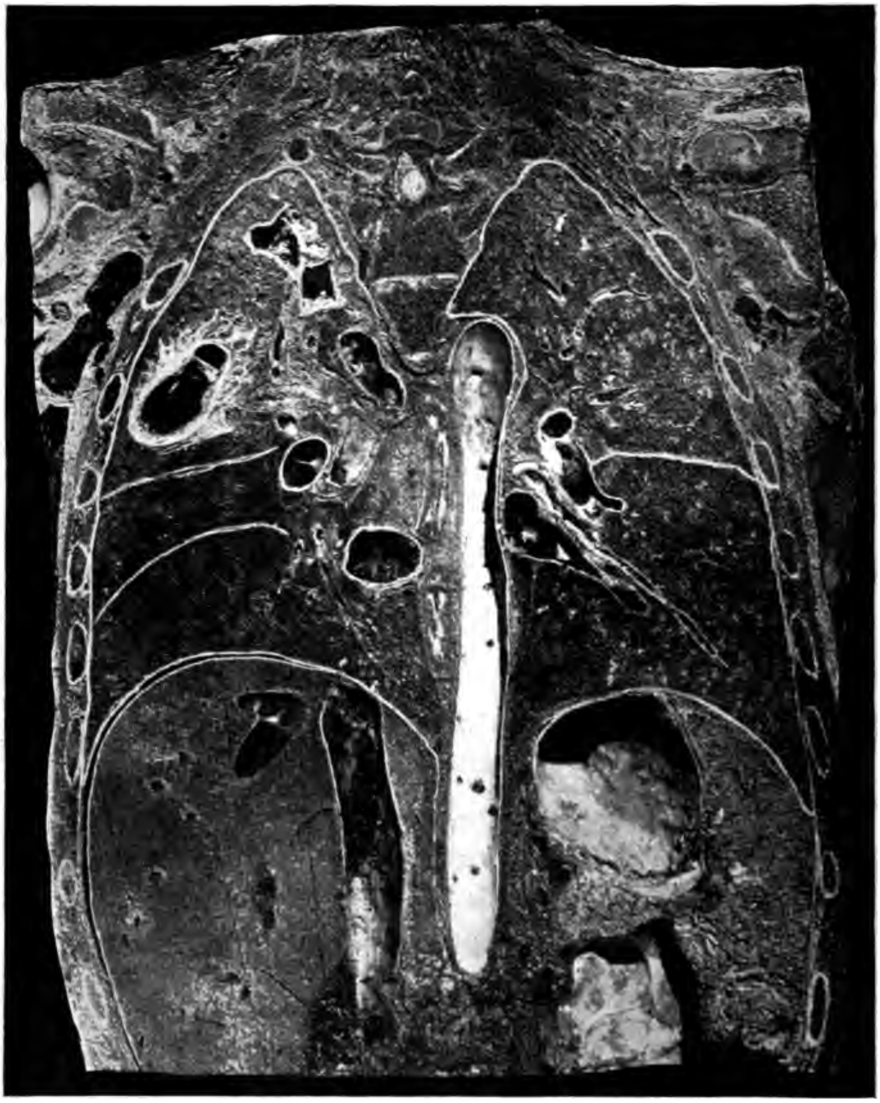

FIG. 61.—Section through the posterior parts of lungs showing *advanced pulmonary tuberculosis.*

The *right upper lobe* shows several empty cavities, one of which is superficial, large, and surrounded by stiff fibrous walls. High up in the axillary region one should expect to find metallic tympany; amphoric breathing; clear-cut, metallic râles; whispered pectoriloquy, and if the bronchus leading into the cavity is stenotic, cracked-pot sound.

The *left upper lobe* shows diffuse infiltration which would yield the following physical signs: diminished expansion, dulness, increased vocal fremitus and resonance, bronchovesicular breath sounds, crepitant and crackling râles.

The position of the liver and spleen in relation to the chest wall, lung and stomach is well illustrated.

to lack of vibratability and may be imitated by percussing the inner surface of the tibia. It is the note heard over consolidated lung tissue. It has less volume (wave amplitude) than resonance and does not carry far.

4. **Flatness** is absolute dulness. It is a *very short, high-pitched non-musical note,* with very little carrying power (intensity). It is heard in percussion of viscera such as the heart and the liver in regions not covered by lung tissue and may be exemplified by percussing the thigh. It is the note of liquids and is heard over pleural or pericardial effusions. It is invariably associated with a *marked sense of resistance.*

SPECIAL PERCUSSION SIGNS

Wintrich's Change of Note.—This consists of a change of pitch during percussion, which varies according to whether the mouth be opened or closed, the note being higher in the former, and lower in the latter instance. In a pipe open at one end, any constriction of the opening lowers the pitch of the sound (Bernoulli). It may be demonstrated by percussing the trachea with the mouth open, and closed. This phenomenon may occur pathologically if a pulmonary cavity or a pneumothorax communicates with an open bronchus. It occurs rarely in pneumonia and is to be explained by conduction of the percussion stroke, through the consolidation to a large bronchus. If noted in recumbency only, this may be due to temporary occlusion of the bronchus by fluid. This sign is of but little practical value.

Gerhardt's Change of Note.—This consists of a change of the percussion sound, with a change of the patient's posture, and is dependent upon an alteration in the direction of the long axis of a cavity, which contains both air and fluid. This was formerly explained as resulting from a change in the length of the air columns. This is incorrect since the greatest possible differences in lung cavities are too small to account for such changes in sound. The real explanation must be sought in the change in tension which as a result of the contained fluid, the cavity undergoes (Geigel).

Friedreich's Change of Note.—This phenomenon consists of a lowering of the pitch of percussion note over a cavity during forced inspiration, due to an increased volume of air. It is, therefore, supposed to indicate a patulous, flexible cavity.

Biermer's Change of Note.—This phenomenon is practically the same as Gerhardt's, except that the former described the metallic resonance sometimes heard over a pneumothorax. The pitch is lower on sitting up, due to an increased volume of the pleural cavity, caused by a sagging of the diaphragm owing to the weight of the effusion.

None of the foregoing changes of note are of much practical importance. They are rarely demonstrable, and even when so are often of doubtful significance.

The Lung Reflex.—Local irritation of the skin, by cold, mustard, or continued percussion, may temporarily produce a reflex dilatation of the subjacent lung tissue —a relative emphysema—which may be sufficient to obscure slight degrees of percussion dulness. Thus too-prolonged percussion of a pulmonary apex may temporarily cause the disappearance of a slight impairment of resonance which was readily demonstrable at the beginning of the examination. It should not be forgotten, however, that aural fatigue on the part of the examiner may account for a similar result. When such an occurrence is suspected, the examiner may proceed to some other step in the examination and revert to the examination of the doubtful region after some time has elapsed.

CHAPTER V

ANATOMIC CONSIDERATIONS

The right ribs are longer, the right lung larger (but shorter), the right shoulder often lower and narrower, the right breast is higher and further from the mid-sternum, than the left.

The Lungs.—The apices extend from 1 to 1½ inches (2.5 to 4 cm.) above the clavicles. The right apex is slightly smaller than the left, its conical shape is due to encroachment of the right innominate vein. This vessel, unlike the left, pursues an almost vertical course, and with the superior vena cava, occupies the space which on the left is filled by the anterior, inner margin of the pulmonary apex. In addition to this the subclavian artery pursues a course more anterior and less mesial, to the right apex than the left. The left apex is more dome-like and slightly larger. There is practically no difference in the height of the apices on the two sides.

The percussion note from the right upper apex to the second interspace is slightly higher-pitched, less resonant, and at times has a slightly tympanitic quality because: (1) The right apex is smaller. (2) The superior vena cava lies in front of the inner part of it. (3) The right subclavian artery occupies a more anterior, the left, a more mesial position. These conditions account for the diminished resonance and higher pitch. (4) The right apex lies in immediate contact with the trachea (Figs. 49, 52, 76, 77, 97). This accounts for the tympanitic element and in part for the elevation of pitch. (5) A slight influence—with light percussion—is sometimes exercised by increased thickness of the right pectoral muscles.

SURFACE LANDMARKS, ETC.

THE RIGHT LUNG	THE LEFT LUNG
Turns out at the 6th costo-sternal articulation (sternal line).	Turns out at the 4th costo-sternal articulation.
	Turns out at the 5th costal space.
Crosses the 6th and 7th intercostal space (mid-clavicular line).	Crosses at the 5th costal space.
	Turns out at the 6th costal space.
	Crosses the 6th and 7th costal space.
Reaches 8th rib in axillary line.	Reaches the 8th in the axillary line.
Reaches 10th rib in scapular line.	Reaches the lower border of 10th dorsal vertebræ in scapular line.
Reaches upper border of the 10th dorsal vertebra.	

Louis' angle[1] (the junction of the manubrium with the gladiolus) marks the sternal attachment of the second rib. It is opposite to the

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[1] Louis' angle, not Ludwig's angle. It was not described by Ludwig, the German, but was named after Louis, the great French clinician, Ludwig being the German for Louis. The angle was originally described by Louis as a unilateral prominence of the ribs noted in certain cases of emphysema on the most affected side.

(E. H. GOODMAN: "Historical Note on the So-called Ludwig's Angle or Angle of Louis." *Medical Record*, July 23, 1910.)

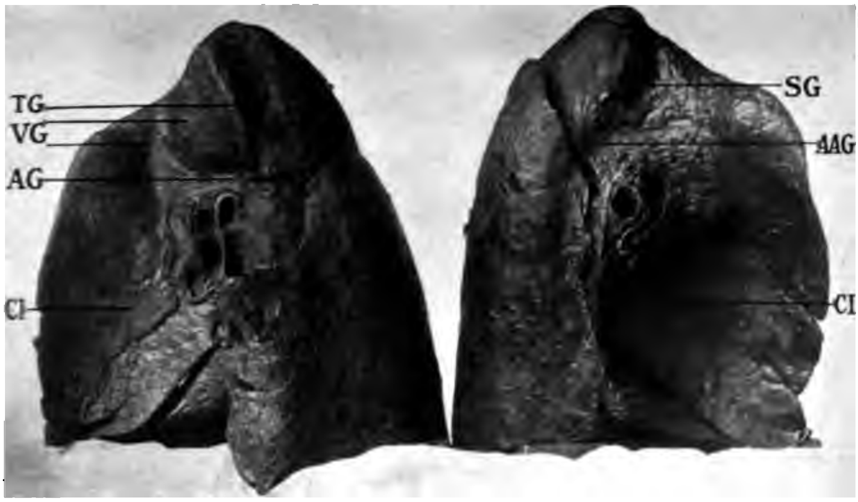

FIG. 62.—Mediastinal surface of lungs hardened before removal. This photograph shows (*a*) the groove produced in the right apex by the trachea; (*b*) the relatively anterior position of the vessels on the right side; and (*c*) the smaller size of the right apex. *AG*, azygos groove, *VG*, grooves for superior vena cava, innominate vein, and subclavian vessels; *TG*, tracheal groove; *SG*, subclavian groove; *AAG*, aortic groove; *CI*, cardiac impression.

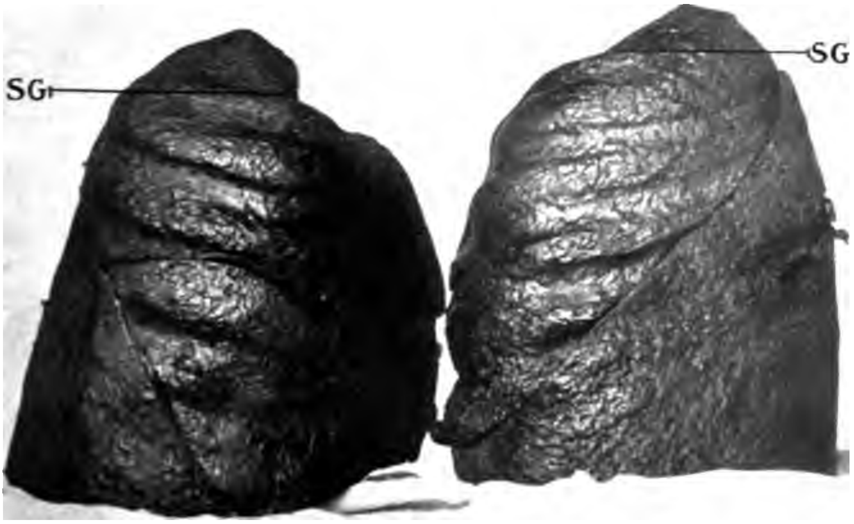

FIG. 63.—Lateral view of lungs hardened before removal. This photograph shows the deeper vascular groove and the smaller size of the right apex as compared with the left.

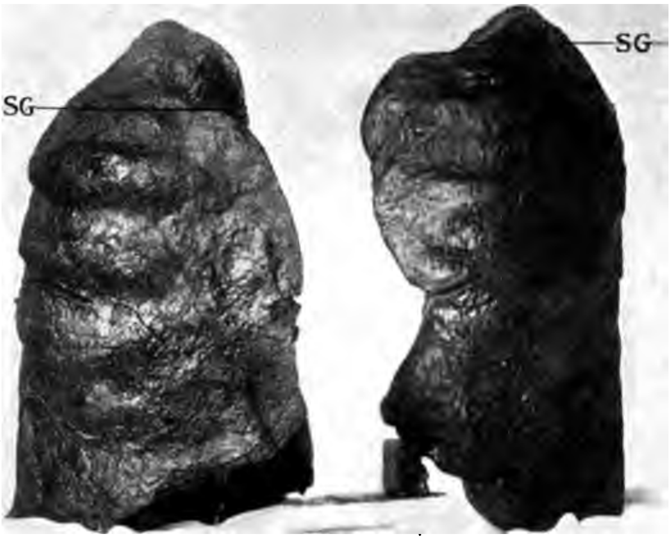

FIG. 64.—Anterior view of lungs hardened before removal. This photograph shows the anterior position of the groove for the subclavian vessels on the right side, compared with the more superior position on the left. *SG*, subclavian groove.

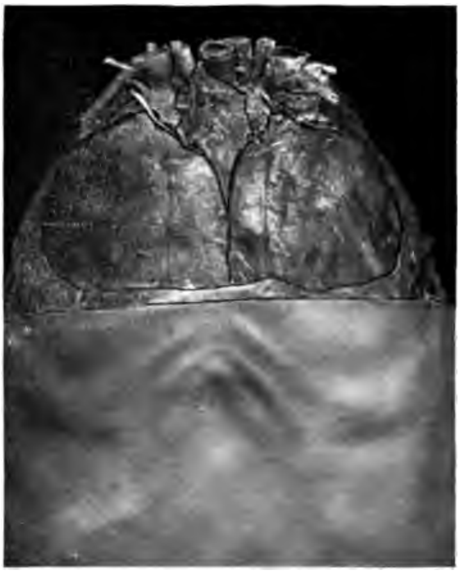

FIG. 65.—The lungs normally meet and frequently overlap at Louis' angle. This previously hardened dissection shows: an infant's chest, displaying a shrunken thymus gland between the apices of the lungs. (*Fetterolf and Gittings.*)

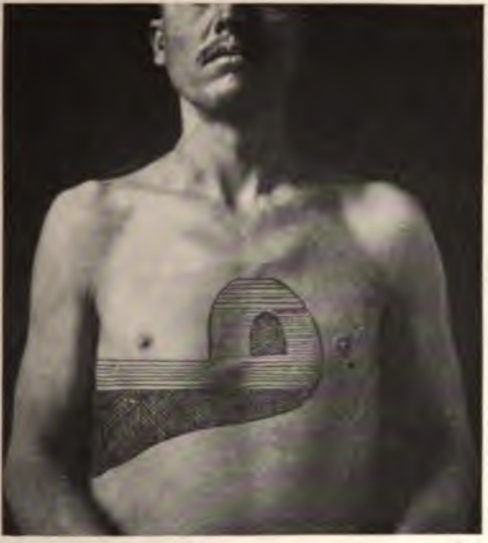

FIG. 66.—Showing prominent angle of Louis, also small areas of absolute cardiac and hepatic dulness in a case of emphysema.

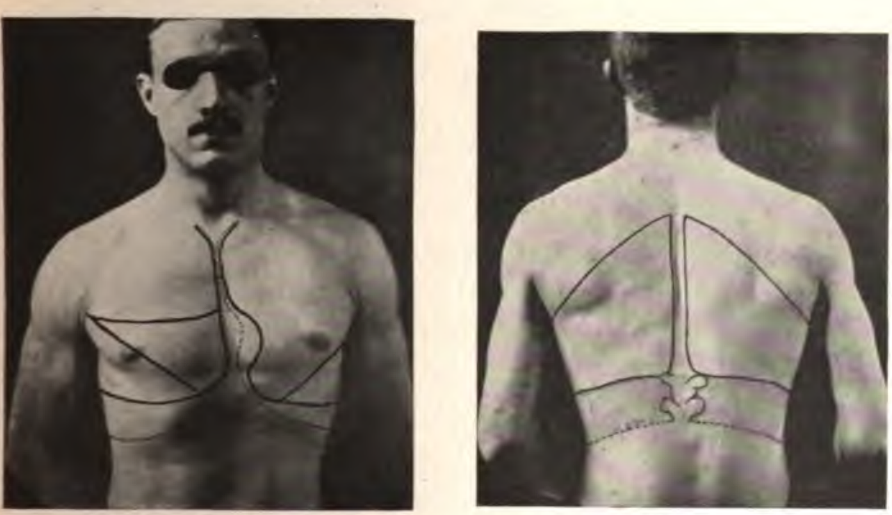

FIG. 67. FIG. 68.

FIGS. 67 AND 68.—The solid lines indicate the position of the pulmonary margins and of the interlobar fissures. The dotted lines mark the anterior and lower confines of the pleural cavities—the spaces into which the lung expands during forced inspiration. This space at its lower margin, which is bounded by the diaphragm on the inside and the thoracic wall on the outside, is known as the *complemental space* of Gerhardt. It is in this space that small pleural effusions first accumulate, thus causing the disappearance of the diaphragmatic shadow (see Fig. 12), slight dulness on percussion and diminished vocal fremitus, resonance and breath sounds.

level of the fifth thoracic vertebra, indicates the level at which the trachea bifurcates, and anteriorly the upper point at which the lungs meet, to diverge again at the fourth costal cartilage. It marks the upper boundary of the cardiac auricles, and the point at which the veins of the hand collapse while the arm is being raised upward from its lowest to its highest position (Gaertner's test of venous blood pressure). It forms a convenient landmark from which to count ribs.

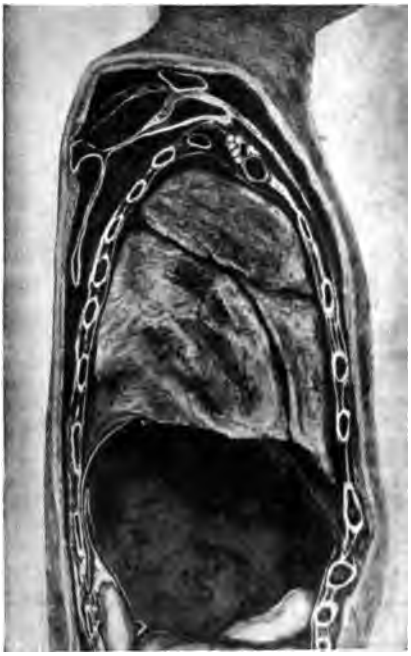

FIG. 69.

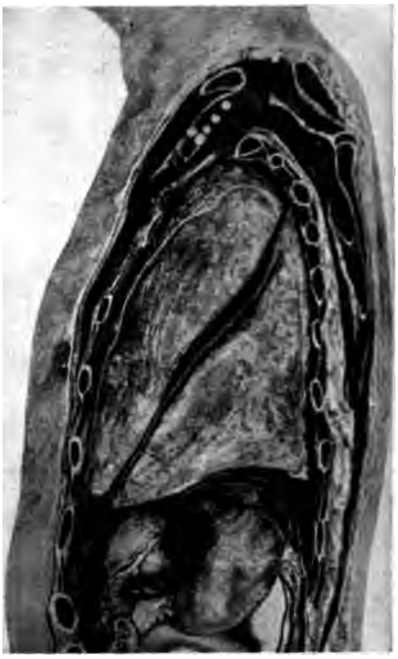

FIG. 70.

FIG. 69.—An approximate knowledge of the location of the pulmonary fissures is especially important in the diagnosis of interlobar empyema. *Right lung.* The fissure between the upper and the lower lobe corresponds to a line drawn from the fourth dorsal vertebra to the fifth or sixth costo-sternal articulation. The upper border of the *middle lobe* is marked by a line drawn from the middle of that just described, to the third costo-sternal articulation. (*After Doyen, Bouchon and Doyen.*)

FIG. 70.—*Left lung.* The interlobar fissure may be located by drawing a line from the fourth dorsal vertebra to the sixth costo-sternal articulation. These relations are not absolutely fixed, but are subject to individual variations. (*After Doyen, Bouchon and Doyen.*)

The *vertebral spines* correspond to the level of the rib below. The first rib begins and is in direct articulation with, the seventh cervical vertebra. The second rib articulates with the second and third vertebræ, the third rib with the third and fourth vertebræ, etc., but the eleventh and twelfth ribs articulate directly with their respective vertebræ. The *scapulæ* overlie the second to the seventh or the third to the eighth ribs. The *hilus of the lung* lies opposite to the spines of the fourth, fifth and sixth, and the bodies of the fifth, sixth and seventh, vertebræ.

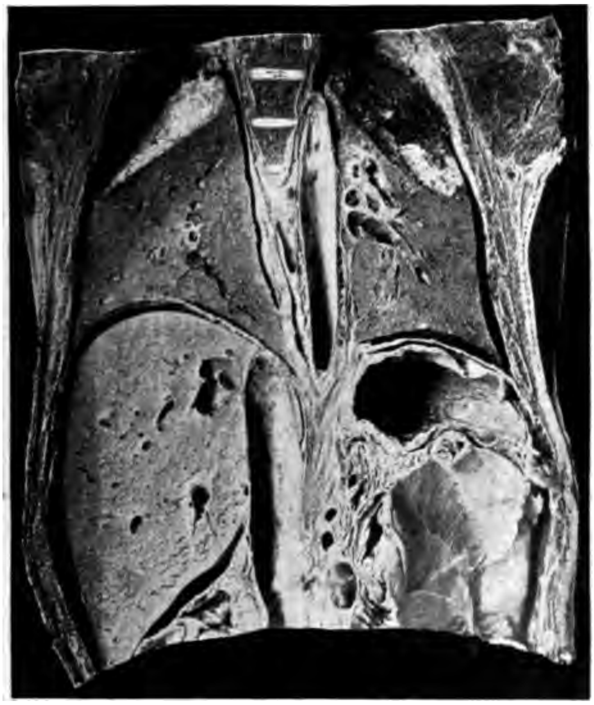

Fig. 71.—Vertical section of the body, showing the shape of the lower lobes, the upper bes having been removed. Note the higher position of the right phrenic dome, which uses the lower border of pulmonary resonance to be slightly higher on the right side. On e left the stomach imparts a tympanitic quality to the lower portion of the lung. If led with food it diminishes pulmonary resonance and may simulate consolidation or a all pleural effusion.

CHAPTER VI

METHODS AND RESULTS OF PERCUSSION

The Immediate or Direct Method.—The hand or the finger tips are struck directly against the object in which we desire to set up vibrations. This was the method of Auenbrugger, who, however, covered the fingers with a soft glove to diminish the finger element of the sound, especially the overtones. The direct method is still occasionally employed to determine the resonance of the upper lobes of the lung (by using the clavicle as the pleximeter), or that of the lower lobes as a whole, by striking the patient's back with the edge of the hand.

The Mediate or Indirect Method.—This was suggested by Piorry, who interposed a hard object—the *pleximeter*—between the *plexor* (the object striking the blow) and the part to be percussed.

Artificial plexors and pleximeters are sometimes used, especially for class demonstration. They generally consist respectively of a small light rubber-tipped hammer, and of a narrow piece of bone or vulcanite of variable shape (Fig. 72). The fingers are nearly always used as plexor and pleximeter.

The middle finger of the left hand is pressed firmly against the chest wall, in a direction parallel to the ribs, while the middle finger of the right hand strikes upon it, just behind the nail, a short, light, quick, vertical blow, delivered from the wrist (Figs. 73 and 74).

The Pleximeter.—Despite the fact that a hard pleximeter, a quick rebound and a short time of impact are advantageous, we cannot use an unpadded plexor of hard material because in such a case a high-pitched sound would be produced, due to the overtones of the pleximeter which would overshadow the low-pitched fundamental note of the lung. We therefore use a soft plexor or the finger, just as we use felt pads in a piano in order to let the basic note preponderate and to drown out the unharmonious overtones. The vibrating area equals in size the percussed area plus a radiation during the course of transit. Hence the smaller the pleximeter and the more superficial the tissue percussed, the more accurate our topographic results (see Fig. 75).

Half the diameter of the pleximeter must always be allowed as the unavoidable margin of error in estimating the boundaries between an air-containing and an airless structure as the heart and lung, even when the pleximeter is applied in a direction parallel to the anticipated boundary line. Based upon these facts some very narrow, wedge-shaped pleximeters (Ziemmsen) have been devised for limiting the lung apices, cardiac boundaries, etc. The results derived from their employment, however, have been disappointing (Fig. 72).

The percussion blow may be delivered either lightly or forcibly, and accordingly we speak of *light* or *heavy*—*superficial* or *deep*—*percussion*. The latter terms are used because with heavy percussion a deeper penetration of the vibrations is ensured—the sphere of the blow is larger—greater masses of tissue are set in vibration and a louder sound is produced.

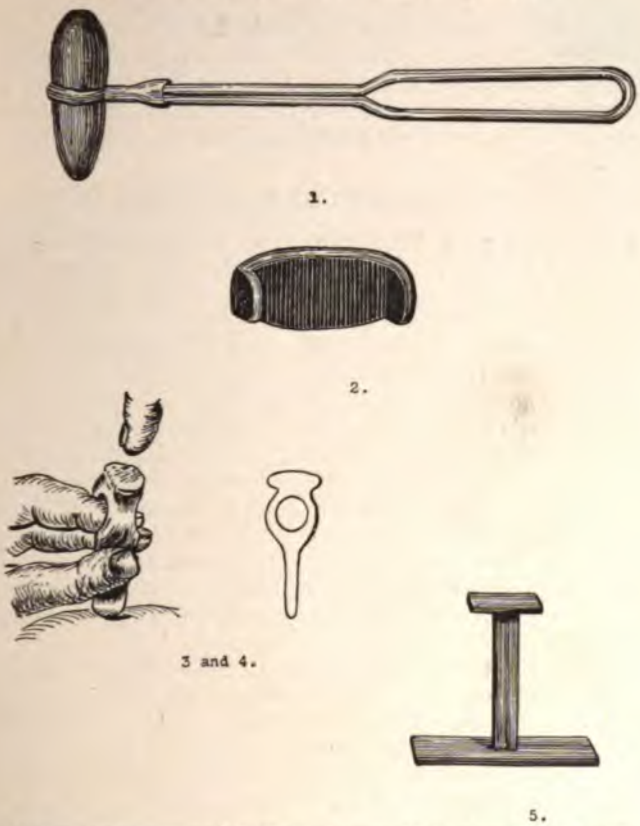

Fig. 72.—1, Plexor or percussion hammer; 2, ivory pleximeter; 3 and 4, Hirschfelder's pleximeter; 5, Sansom's pleximeter.

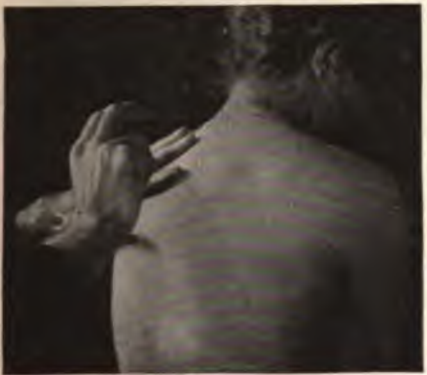

Fig. 73.

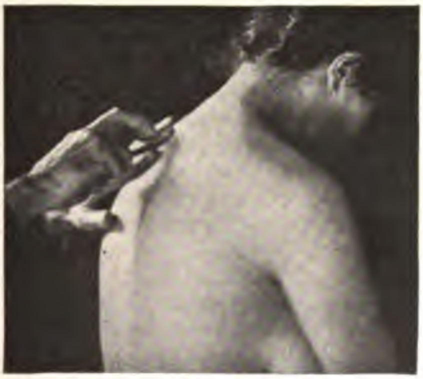

Fig. 74.

Figs. 73 and 74.—The method of percussion. The percussion blow is struck from the wrist only, the forearm being practically stationary. The impact, which is delivered just behind the nail of the middle finger, should be quick and brief in duration, the force of the blow should fall as vertically as possible. Fig. 73 shows the beginning, Fig. 74 the end of the percussion stroke. In order to deliver a vertical blow with the terminal phalanx, the nail of the second finger, which is generally used as plexor, must be short.

The fingers are preferable to artificial plexors and pleximeters because of the important information derived from the *sense of resistance.* It is a relatively easy task, for one practised in percussion, to outline accurately dull areas by this means alone. It is a useful method when we are forced to make our examination in the presence of extraneous noise.

THE RESULTS OF PERCUSSION

As the result of a percussion stroke on the chest wall, the equilibrium of the underlying tissues is locally disturbed, they are thrown into vibration, and a sound results. This sound is produced by vibrations arising from three sources:

1. *From the Pleximeter.*—This element of the composite sound is high-pitched, of short duration and dull.

2. *From the Thoracic Wall.*—This element also yields a short, dull, high-pitched (osteal) note. When the costal cartilages are ossified much of the percussion blow is absorbed by the arch-like structure of the ribs, penetration is diminished, and a more or less tympanitic note is produced.

3. *From the Underlying Tissues* (Lungs, etc.).—That portion of the sound which is contributed by the vibrations of a healthy lung under normal tension is long, low-pitched and resonant, because of the thinness and elasticity of the pulmonary tissue, and the large amount of air it contains. The greater the depth of the lung, the lower the note.

Sound travels radially from point struck and gradually fades. It is conducted through tubes (bronchii, stethoscope) to much greater distances because lateral radiation is minimized.

Sound waves in traveling through the body meet with media of varying density and are in part (*a*) reflected; (*b*) transmitted (by setting up vibrations in the second medium). *In passing through media of different densities much sound is lost by reflection.*

In percussing the chest the overlying tissues in different symmetrical regions are much the same (skin, fat, bone, etc.) and, therefore, comparisons of the underlying tissues can readily be made. But when fat or edema are excessive, percussion data are obtained with difficulty.

Sound is conducted better if its waves impinge vertically upon the dividing point of two media than if they reach it obliquely. Hence *the percussion stroke must fall vertically* in order to penetrate deeply.

Although percussion sets up vibrations in the entire lung, yet it does so in the deeper portions insufficiently to set up audible sounds. Practically, therefore, in case of a *normally distended lung with light or moderate percussion, the vibrations tend to remain* localized both as regards lateral radiation and vertical penetration.

It has been shown orthodiagraphically that the heart may be accurately outlined both by forcible and by very light percussion. It was questioned whether threshold percussion had sufficient penetration to traverse the lung overlying the heart or whether the alterations of sound were not simply the result of altered pulmonary tension due to the nearby solid organ. Weil's statement made years before and generally accepted was to the effect that percussion vibrations penetrated only 5 to 7 cm., thus only 4 to 5 cm. into the lung itself. This belief was founded on the fact that liver dulness could not be demonstrated when more than 4 to 5 cm. of lung tissue overlay it. The following experiments show that even light percussion has a deep penetration.

I. Moritz and Rihl beat up into a spongy froth, a solution of stiffening gelatin to which formalin and carbolic acid had been added, this closely resembling lung tissue in ture. This was poured into a glass cylinder 4 cm. in width and 70 cm. in length. re the gelatin had "set" a small rubber balloon was placed in its midst, not in act with the glass, and connected by means of a tube with a sensitive flame. The est possible percussion produced vibrations in the flame.

I. A cylinder 20 by 20 cm. gave the same tone whether light or heavy percussion used, showing that in either case the same mass of air vibrated.

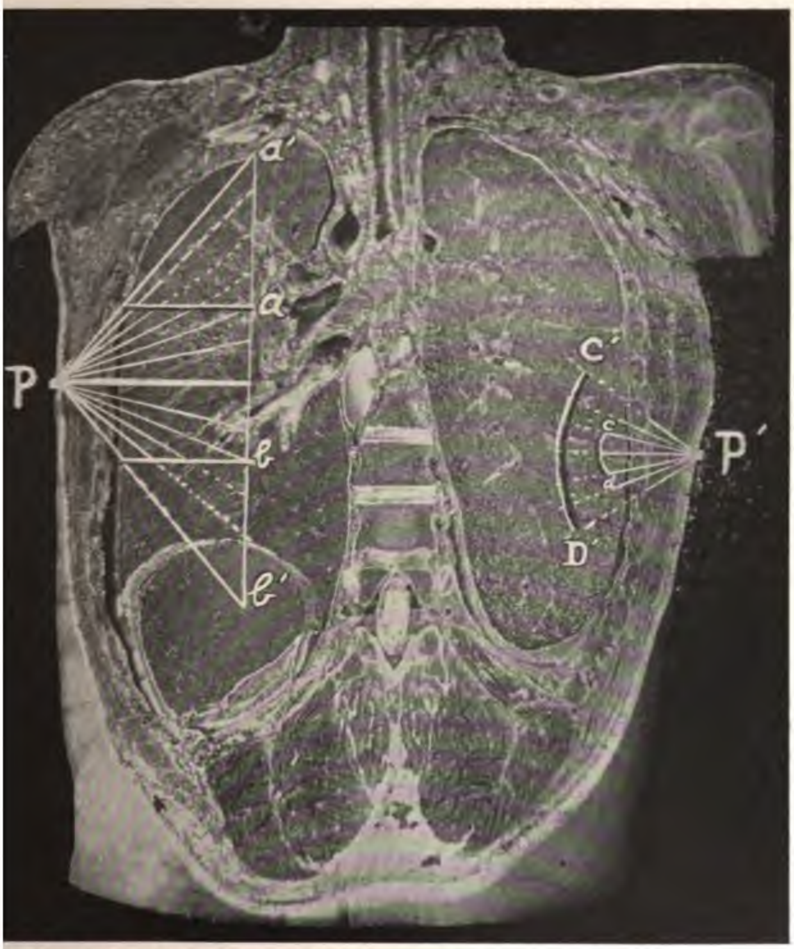

FIG. 75.—Diagram illustrating the penetration of audible percussion.

Very heavy—3 finger or fist *percussion*—at *P* produces audible vibrations throughout the triangle This tends to bring out the note of the lung as a whole.
Heavy percussion at *P* sets up audible vibrations throughout the triangle *aPb*.
Average percussion at *P'* sets up audible vibrations throughout the area *C'P'D'*.
Very light percussion at *P'* causes audible vibrations only throughout the area *CP'D*.

II. Wintrich showed that percussion of the clavicles in the corpse was readily ible by the hand held beneath the diaphragm. Here, of course, bony conduction occurred.

V. If lung tissue be immersed in water whose surface is percussed, the pulmonary ance can be elicited if percussion be performed directly over the lung, but not if troke is delivered beyond its margin. But, of course, percussion vibrations are more directly conducted by water than by air or lung tissue, especially when in case of the latter the bony thoracic arch is interposed.

V. Moritz and Rihl further showed by modifications of the gelatin experiment and by attention to physical laws such as that regarding the parallelogram of force, that the laterally radiating percussion waves in media denser than air lose much of their penetration power.

An ovoid cylinder yields a lower note when percussed in the direction of its long axis.

It is evident, therefore, that the physical character of lung tissue is such that a vibration of the entire mass always occurs even with the lightest percussion. Nevertheless it reacts to percussion in such a manner that a louder note is produced when a larger mass of pulmonary tissue is directly exposed to the impact than when only a thin layer of lung tissue underlies the area of the percussion blow.[1]

The following points, therefore, deserve to be emphasized. *Light percussion is essential for topographic diagnosis; with heavy percussion too much lateral radiation occurs. Heavier percussion must be used where the superficial tissues are thick, in order to set up audible pulmonary vibrations, but at the same time the accurate delimitation becomes more difficult. Very heavy percussion may give us a general idea as to the amount of air-bearing tissue in the lung as a whole. It is absolutely useless for the purpose of outlining of organs or for the detection of small areas of pulmonary infiltration.*

For practical purposes then, in so far as audible sound production is concerned, the *penetration of the percussion wave* is about 6 cm. (2¾ inches). Of this, 2 to 3 cm. is consumed in penetrating overlying tissues so that only 3 or 4 cm. (1½ inches) are left to enter the underlying organ. Hence percussion is practised from all directions. The heart could not be outlined from behind, nor deeply placed pulmonary lesions from in front. The fact, however, that consolidations the size of a cherry, and deeper than 2¾ inches cannot be located by light percussion does not prove that sound waves may not penetrate deeper. Deep percussion tends to bring out note of lung in its entirety, and drowns out slight degrees of dulness.

For *light percussion* the pleximeter must be lightly but snugly applied to the chest wall, the stroke must be gentle and its duration short, so as to include only a small sphere of blow (Fig. 75). We should percuss so as to evoke practically no note over the dull areas. Light percussion is more accurate because with it there is less lateral radiation. For *deep percussion* the plexor is applied more firmly, the blow is somewhat longer and more intense. Too strong a blow is transmitted too far laterally and renders exact localization impossible. Deep percussion is not necessarily loud percussion. *Percussion should be of such strength and duration, as to make the difference between resonance and dulness as great as possible.*

"The deep dulness of organs depends only on the volume of the air-containing parts in the region of the sphere of the blow" (Weil) and not as was formerly taught upon the dulling influence of the neighboring solid organs upon those filled with air. Such a dulling influence does not exist (Sahli). The strength of percussion must vary with the size and character of the organs and neighboring tissues: *e.g.*, in children (thin chest walls, superficial organs) light percussion is necessary; in corpulent adults (thick chest walls, deeply placed organs) heavier percussion is required.

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[1] MORITZ and RIHL: *Deut. Arch. f. kl. Med.*, 1909.

As has been stated, the percussion note obtained over the lung is a compound sound, consisting of the note of the pleximeter, the chest wall, and lung. In heavy percussion it is the lower, and in light percussion the higher, range of this complex to which our chief acoustic attention is directed. Both have the higher range—the pleximeter note—in common, but there is relatively more of the pleximeter note in the sound produced by light percussion. Lessening of the amount of pulmonary air is manifested by a lack of the deeper tones.

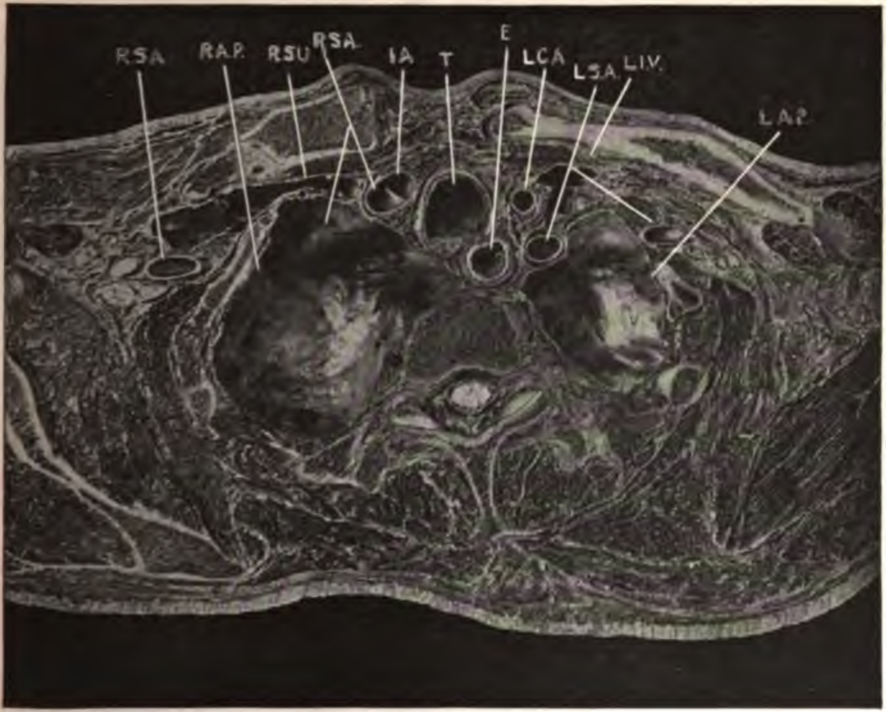

FIG. 76.—Section through the upper part of the thorax, viewed from below. The line of section is not exactly horizontal, a slightly lower plane being reached on the right side than on the left. In order to show the apical parietal pleura, the pulmonary apices have been removed. There can be noted in this specimen the beginning contact of the right pleura with the trachea, and the anterior position of the innominate artery, whose bifurcation is well shown. On the left side, the wide separation of the pleura from the trachea by means of the large arteries, esophagus, and areolar tissue can readily be seen. The deep position of origin and the obliquely anterior course of the left subclavian artery is plainly visible. *T*, trachea; *E*, esophagus; *R.A.P.*, right apical pleura; *L.A.P.*, left apical pleura; *I.A.*, innominate artery, dividing into *R.S.A.*, right subclavian artery and *R.C.A.*, right common carotid artery; *L.S.A.*, left subclavian artery; *L.C.A.*, left common carotid artery; *R.S.V.*, right subclavian vein; *L.I.V.*, left innominate vein.

If one lung becomes infiltrated, its percussion note becomes higher in pitch, not by virtue of any new sound element added, but on account of the loss of the deeper tones. The note of consolidation is, therefore, to speak accurately, not actually higher but relatively less low. It is the prominence of the low-pitched notes which causes what we know as resonance. The difference between a resonant and a dull note lies, however, not only in the rate of the vibrations (pitch) but also to some extent in their amplitude (intensity). In other words, the resonant note is louder and carries further. For this reason slight degrees of dulness can sometimes be more readily perceived at a slight distance from the patient than by the percussor himself. This is due to the fact that sound waves lose much of their amplitude in transit, and the difference between 1 and 0 seems greater than that between 1 and 2. Low-pitched sounds having a great vibratory excursion carry further. In listening to a distant orchestra we hear the drums and bassoons, not the stringed instruments or the fifes; we hear the distant pounding of the surf, not the swish of the waves.[1]

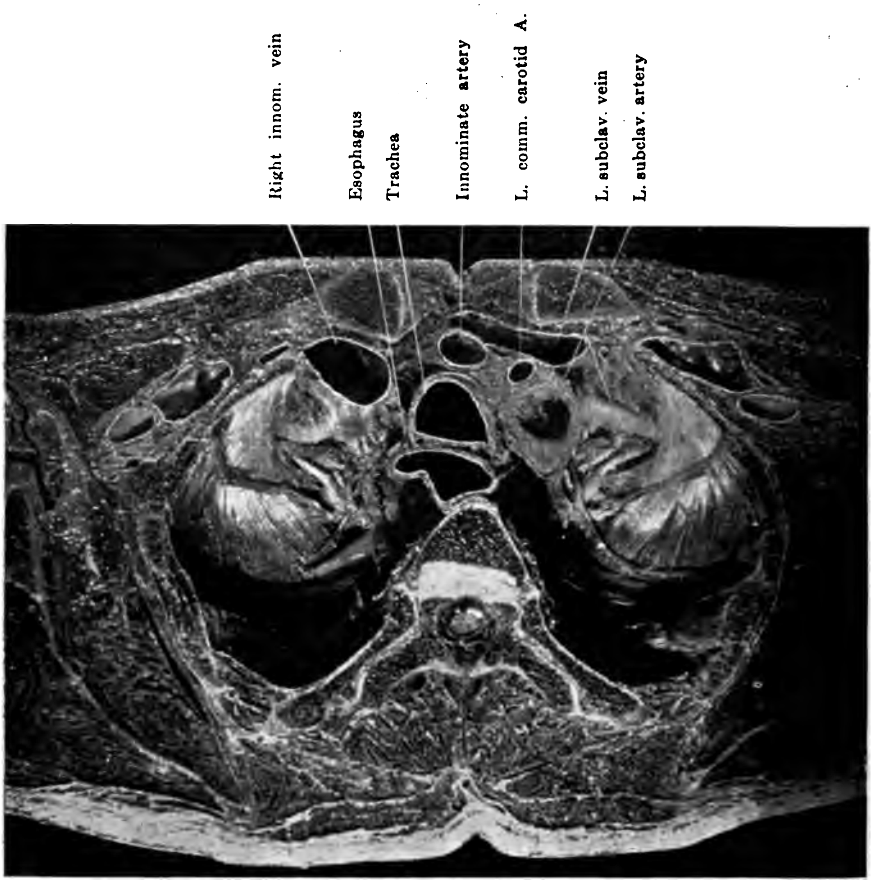

Fig. 77.—Horizontal section of the thorax just above the sternoclavicular articulation viewed from beneath. The pleura has been dissected away. This photograph shows (*a*) the anterior position of the innominate artery, (*b*) the posterior position of the left subclavian artery, and (*c*) the anterior position of the right innominate vein. *R*, first rib; *R.I.V.*, right innominate vein; *E*, esophagus; *T*, trachea; *I.A.*, innominate artery; *L.S.V.*, left subclavian vein; *L.C.A.*, left common carotid artery; *L.S.A.*, left subclavian artery.

It also shows the thickness of the dorsal muscles through which the percussion vibrations must penetrate in order to set up vibrations in the lung, even above the level of the scapulæ. Percussion must, therefore, be more forcible than on the front of the chest. This section, although from a different body, shows the same anatomic disposition of the large vessels in relation to the upper lobes as does Fig. 76. It is in part due to the more anterior position of the vessels in case of the right upper lobe that the percussion note on this side is normally less resonant than that of its fellow.

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[1] Selling: *Deut. Arch. f. kl. Med.*, vol. xc.

The skilled examiner can be accurate in topographic percussion within a range of from 0.5 to 1.0 cm. Whichever method we employ, the finger which strikes the blow (plexor) must fall vertically, not obliquely, upon the tissues to be percussed for thus greater penetration is ensured. Furthermore, it must be quickly withdrawn, for thus lateral deviation and radiation of vibration is minimized, and deeper penetration ensured. In other words, unrhythmic vibrations are produced, in contradistinction to rhythmic ones, in which the lung tends to vibrate as a whole, and in which topographic delimitation is impossible.

THE PURPOSE OF PERCUSSION

Percussion is practised: (1) *to elicit sound;* (2) *to determine resistance.*

Percussion is based upon the fact that when the equilibrium of an elastic body is briefly disturbed, it is thrown into vibrations, which in turn produce sounds which vary in *intensity*, *quality*, *duration* and *pitch*, such variations depending on the character of the vibrating tissue—the amount of air and the degree of elasticity—and on the nature of the blow.

The bodily organs are of unhomogeneous structure and for the most part not good tone producers, but sounds can be produced in air-containing chambers and in liquid-containing organs. The former vibrate more easily and for a longer time.

The vibratory capacity of tissues depends upon:

1. *The size and character of the air-containing spaces*—lungs, intestines, etc.

2. *The amount of elastic tissue.* The skin and the lung have a good deal, the liver little. In the lungs it is under tension, hence a better tone than that of the skin.

3. *The degree of tension.* The greater the tension, the higher the pitch —tympanitic belly. Human tissues—the lung, intestines, etc.—vary greatly in the degree of tension they possess. They may be so relaxed as to yield no note; or they may be so tense as to yield metallic sounds.

4. *The thickness of the tissue.* The thinner the tissue, the better the vibration, the higher the note.

Various tissues vibrate differently, and the same tissues when altered by disease in their physical consistencies yield an altered note. Percussion teaches us only *physical differences.* Before we can make a diagnosis we must add the data obtained by inspection, palpation, auscultation, etc., as well as the knowledge we possess regarding the history and symptoms of the patient and the pathology of the disease.

For example, a dull note obtained over lung tissue teaches us that the tissues are less vibratile—contain less air—than is normally the case. The sound of the pleximeter being constant, we are justified in attributing variations in the sound to physical alterations in the underlying tissues. Such a dull note generally indicates consolidation, but even if we are correct in this assumption we have no means of determining by percussion with which of the diverse types of pulmonary consolidation we are dealing.

Percussion is also used *to outline the boundaries of organs* when neighboring viscera possess different physical qualities, as for instance the heart and the lung. The most brilliant triumphs of topographic percussion are manifested when unrhythmic vibrations (which remain more or less localized) are set up, and when the tissues directly underlying the point of percussion yield different vibrations than the neighboring structures, *i.e.*, in the normally distended healthy lung. We can accurately outline the heart or the liver from the lung since they have very different vibratory qualities, but not the liver from the heart, since their acoustic properties are much the same. Neither can we be accurate in differentiating between the stomach and the colon, owing to their vibratory similarity, nor can we accomplish much in outlining a tumor of the intestine because rhythmic vibrations (tympanitic sounds) are set up, which cannot be localized.

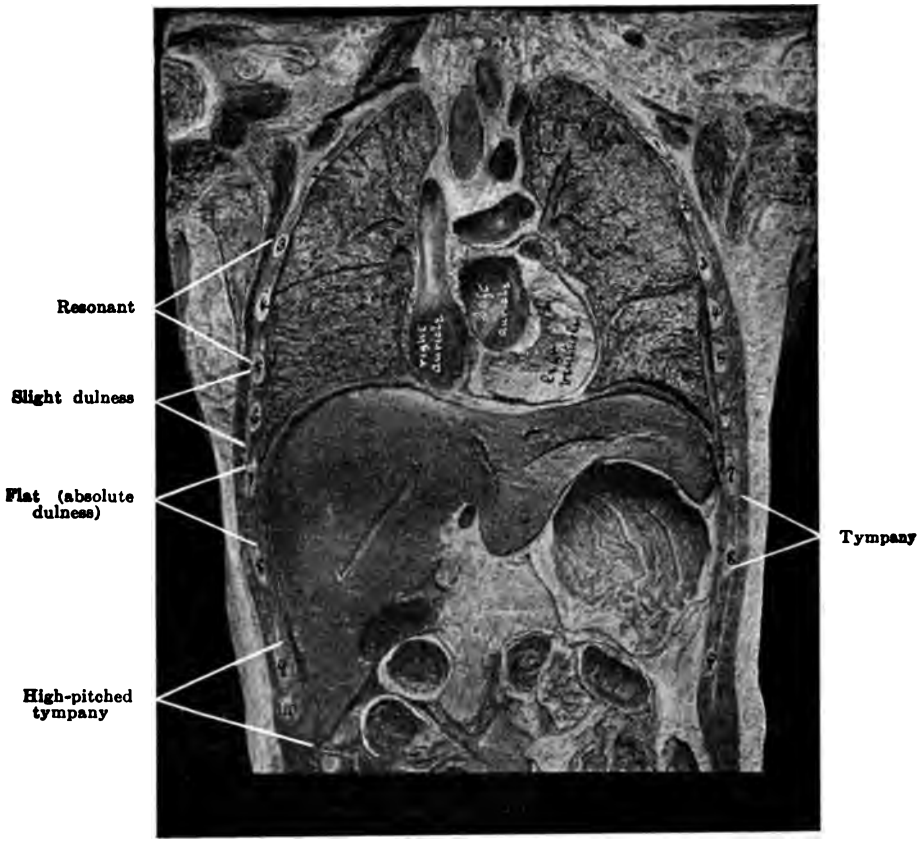

FIG. 78.—Illustrating the different percussion sounds normally obtained on the right anterior and the lateral aspects of the chest, as well as the anatomic reasons therefor. (Compare Fig. 88.)

The Sense of Resistance.—Hard and soft are terms which we apply to objects according as their parts are displaceable with difficulty or with ease. We derive our judgments from the pressure sense of the skin and the muscle sense. Thus we speak of a board-like resistance, which we are accustomed to meet with in massive consolidations.

The attributes which give us the impression of hardness or softness depend essentially, *first*, upon the elasticity of the percussed object. The more elasticity, the greater the displacement possible. *Second*, upon the length of time during which the percussed object is out of contact. The longer the plexor and pleximeter remain in contact the softer the object seems and *vice versa*. Now we noted under the discussion of tympany that heavy and prolonged percussion strokes were unfavorable to bringing out unrhythmic vibrations—non-tympanitic notes. And we find that an increased tactile impression is most common in association with dulness. A sense of increased resistance is not compatible with pure tympany; the note which goes hand in hand with such increased resistance is non-tympanitic, of slight intensity, of brief duration, and high in pitch (flatness). Thus pulmonary consolidation and more especially liquid exudates, as a pleural effusion, produce a marked increase in resistance which is readily felt by the underlying (pleximeter) finger, and which is associated with a short, high-pitched, flat percussion note.

Not infrequently we are able to state positively whether a given note is either resonant or flat, even with our ears closed, simply by our sense of touch. Anything which diminishes elasticity will increase resistance.

THE TECHNIC OF PERCUSSION

The patient may be sitting, standing or lying, the preference being in the order named. The chest must be exposed or covered only by an examining cape (see p. 21). Muscular relaxation must be secured. A revolving low-backed chair or stool is useful, since it renders change of position from front to back easy and expeditious.

The examiner proceeds to percuss alternately corresponding areas of the right and left sides of the body and to note differences in the sound and resistance encountered. Many of these differences are due to normal anatomic causes and these must of course, be borne in mind.

Although it is generally the custom to begin at the top and percuss to the bottom of the chest, this method is, as has been pointed out by N. K. Wood, especially for beginners, much more difficult, and for the following reasons more fraught with the possibility of error.

It is much easier for the ear to single out a high note from among low ones, than is the reverse. In tuberculosis especially, it is the upper part of the lung which is first and most extensively diseased. By beginning at the base, therefore, we are more likely to have a normal resonance and resistance to begin with. In other words, it is better to progress from a normal finding to pathologic change, than work from the abnormal to the normal. This applies to both the percussion sound and to the sense of resistance.

The *upper border of the liver* may be very accurately determined by marking on the chest wall a line at the point at which, while percussing downward, the first slight impairment of resonance occurs. Percussion is then begun from the costal margin upward and the point marked at which with threshold percussion an audible sound is produced. X-ray studies have shown that the upper hepatic border corresponds to a point midway between the two lines (Laporte). The lower border may better be determined by palpation since tympany due to the hollow viscera renders localization of vibrations impossible.

Percussion and auscultation of the *apex of the axilla* will often reveal physical signs which may be sought in vain elsewhere. This is especially the case in the early stages of pneumonia, in pulmonary tuberculosis and in interlobar empyema. The accompanying illustration (Fig. 79) depicts the position in which the patient's arm should be held for such an examination. The muscles being retracted examination is facilitated.

SPECIAL VARIETIES OF PERCUSSION

Spinal Percussion.—Percussion of the spine while not generally practised, is believed by some physicians to have a value in the diagnosis of deep-seated aneurisms and tumors, mediastinal lymphadenitis, etc. There is normally dulness from the first to the fourth dorsal vertebræ; and an "osteal" note extends thence to the last dorsal spine; the lumbar region yields an impaired note, and the sacral vertebræ, tympany. We have never been able to convince ourselves of the value of this method of examination (see Fig. 91).

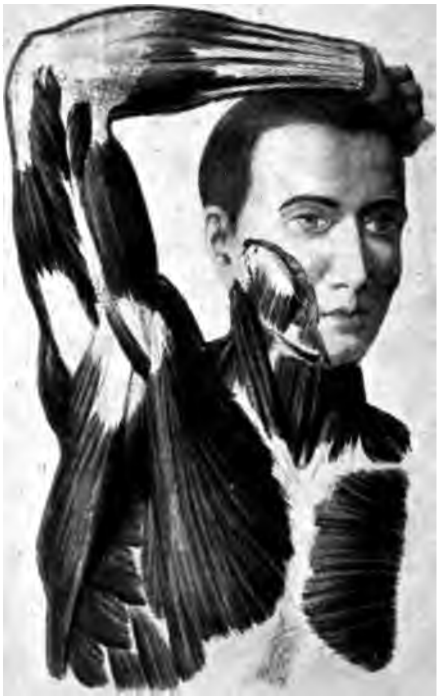

FIG. 79.—Position of the patient while the axillary region is being examined.

Threshold Percussion.—This is simply the lightest of light percussion. It must be done in an absolutely silent room. The pleximeter is laid lightly against the chest wall, only in the interspaces, the blow is delivered upon the proximal end of the second or of the distal, phalanx which is bent at right angles to its fellow, and in a vertical direction upon the chest (orthopercussion). If this is impossible, a stump of lead pencil may be substituted. In other words, the pleximeter is not only small but vertically placed. The blow struck by the plexor is so light that only resonant tissues produce any audible sound; when dull tissues are struck their note is below our auditory threshold (Fig. 80).

The Coin Test.—In performing this test silver coins are used, as the plexor and pleximeter respectively. Percussion is practised over the front of the chest, auscultation over the back. The object of using coins is to favor the genesis of very unrhythmic vibrations (metallic sounds). If a pneumothorax is present, the sounds heard through the stethoscope will have a clear, vibrant, bell-like quality, owing to the resonating quality of a large cavity. In such a case, the coin test is said to be present and the presence of pneumothorax is practically established.

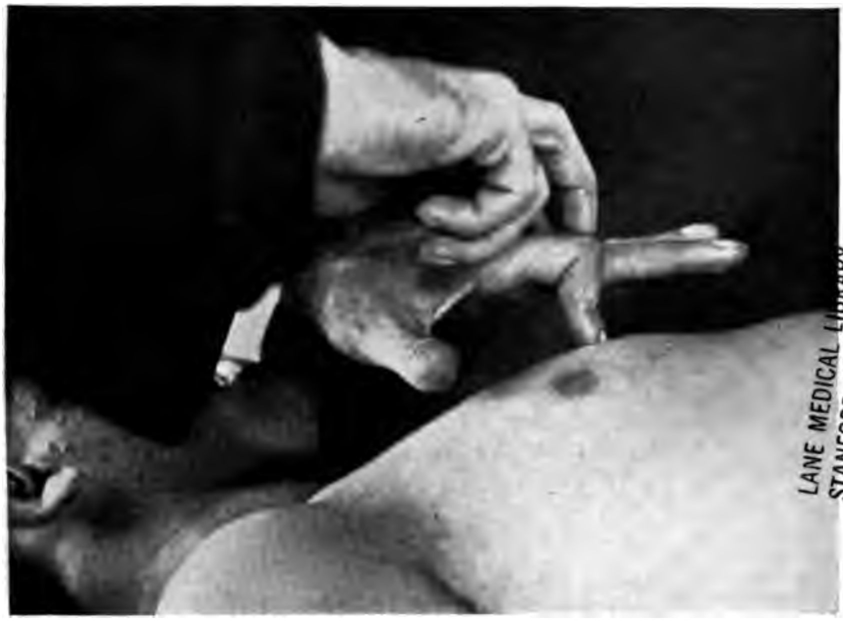

FIG. 80.—Orthopercussion. Generally employed in conjunction with a very light percussion stroke. The pleximeter surface being small and the blow vertical, great accuracy may be obtained. This method is especially recommended for outlining the left border of the heart.

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Normal lung, solidified lung and simple effusions yield only a dull, short metallic note closely similar to that heard over the healthy lung (see Fig. 60).

Auscultatory Percussion.—This is occasionally of use in outlining organs. The bell of the stethoscope is placed over, let us say, the heart. It is held in position by the patient or an assistant while the examiner percusses *lightly* the surrounding regions in a circular direction.

We begin at a distance from the heart and percuss toward it in a series of circles. The tissues, the sound of which is to be compared, must be equally distant from the stethoscope, as under normal conditions the sound will increase in intensity in approaching the point ausculted. As soon as the organ is reached by the percussion blow the sound will greatly increase in intensity and we know that its outer boundary has been reached. Instead of percussing some examiners use a vibrating *tuning fork*, placing the butt of the same against the chest wall in order to set up intrathoracic vibrations.

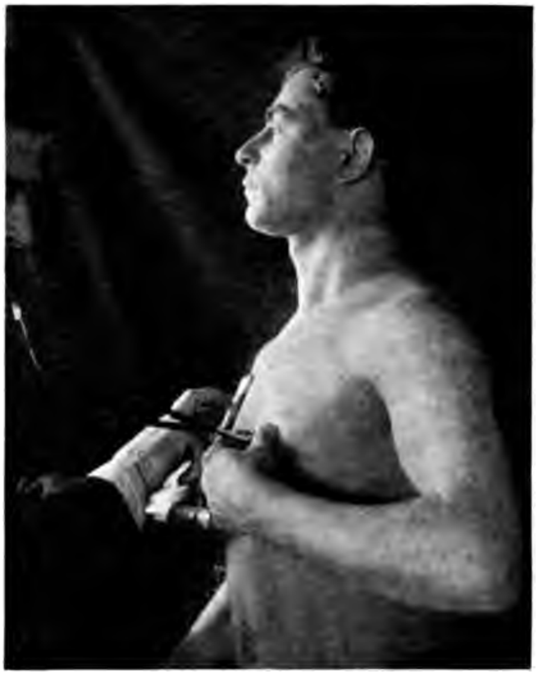


FIG. 81.—Auscultatory percussion.

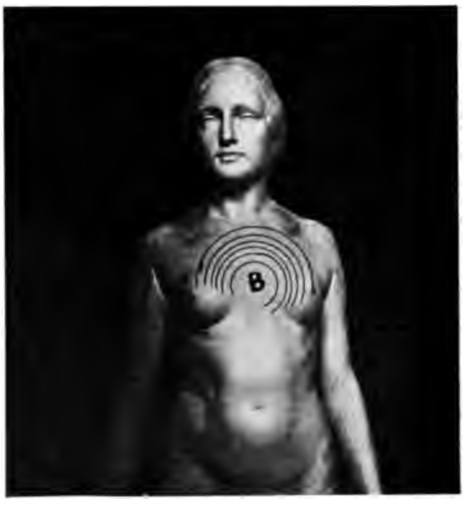


FIG. 82.—Auscultatory percussion. The bell of the stethoscope is placed at B and percussion is performed in gradually narrowing circles toward the heart.

CONDITIONS MODIFYING PERCUSSION SOUNDS

1. Any thickening of the superficial tissues tends to diminish penetration of the percussion blow and to deaden sound. The commonest causes are: adiposity, large muscles, or mammary glands and edema.

2. When the bony thorax is large and deep, we have a more powerful resonator. This tends to cause a note of lower pitch. When the costal cartilages are ossified, the thorax becomes more rigid, and less penetration of the percussion blow occurs because a greater portion of the impact is transferred laterally by the arch-like structure of the ribs, accurate topographic percussion is, therefore, more difficult.

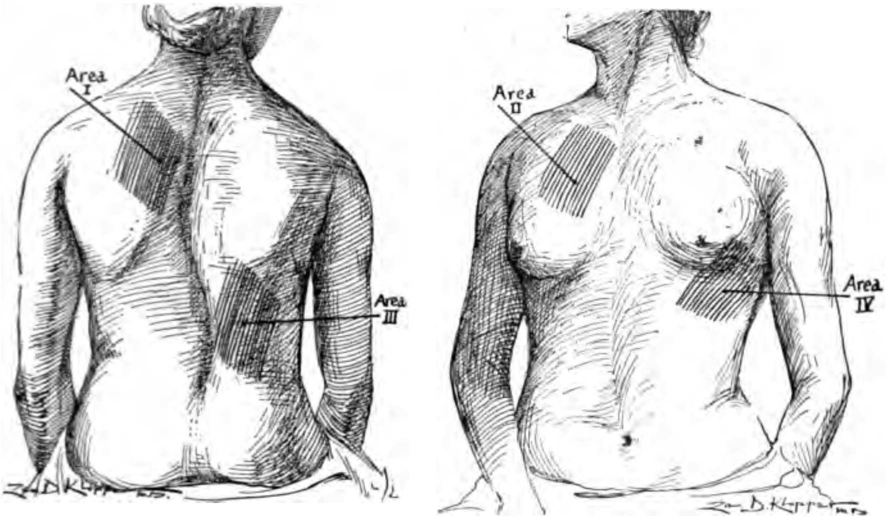


FIG. 83. FIG. 84.

FIGS. 83 AND 84.—Showing the areas over which the percussion note is shorter, higher pitched and less resonant than normal in dextro-convex scoliosis. These changes are most marked over the area I, and least marked over the area IV. (*After E. A. Gray, Jour. Am. Med. Assn.*, 1912, LIX, 2249.) (See Figs. 17, 34, 36.)

3. Thickening of the pleura, or exudation or effusion into the pleural cavity cause both a diminished penetration of the percussion blow and a lessened resonance of sound, because the vibrations lose much of their amplitude in passing through media of different density.

4. Changes in the pulmonary tissue—the degree of tension, the amount of air, elasticity and density—produce marked and important alterations of sound.

I. Changes in pulmonary tension may be uni- or bilateral.

(*a*) *Increased tension* occurs during forced inspiration and in emphysema. This causes more rapid vibrations and a higher-pitched note.

(*b*) *Decreased tension.*

(1) *General,* may arise from pleural effusions, increased intra-abdominal pressure (ascites, tumors); it also occurs in senility (less elastic and more rigid thorax). Decreased tension allows the lung to vibrate as a whole. This adds a tympanitic quality to the percussion note.

The high-pitched tympany which occurs above pleural effusions and around consolidations is known as *Skodaic tympany* (Figs. 107, 320).

(2) *Local* decrease of tension occurs around consolidated areas (pneumonia, tuberculosis, etc.).

II. Changes in the Amount of Air in Lung Tissue.—(*a*) An *increase* occurs in emphysema, asthma and during compensatory (forced) breathing, also in cases of cavitation. In the first case the percussion note is hyperresonant, in the last, tympanitic, provided that the cavity is sufficiently large, not too deeply placed, and that it is filled with air. In the first instance the vibrating tissues are thinner under greater tension and contain more air. In the latter the walls of the cavity vibrate in unison with the air columns it contains. During forced inspiration deep percussion yields a lower; light percussion, a higher note, than during forced expiration.

(*b*) *Decreased* air bearing lung tissue occurs in consolidation (pneumonia, tuberculosis, fibrosis, atelectasis with compression, cavities filled with exudate, etc.). The percussion note becomes less resonant in proportion to the consolidation, because the vibrating tissues are thicker, less elastic and contain less air. The sound, therefore, becomes short, high-pitched and dull, and resistance is increased.

Even minor degrees of *spinal curvature* may produce lack of resonance over certain areas of the chest. This is due to the fact that abnormal convexities of the thorax produce an increased rigidity of the more convex rib which tends to prevent penetration of the percussion blow, whereas a flattened rib has the opposite effect (Sahli) (Figs. 17, 26, 36, 83, 84).

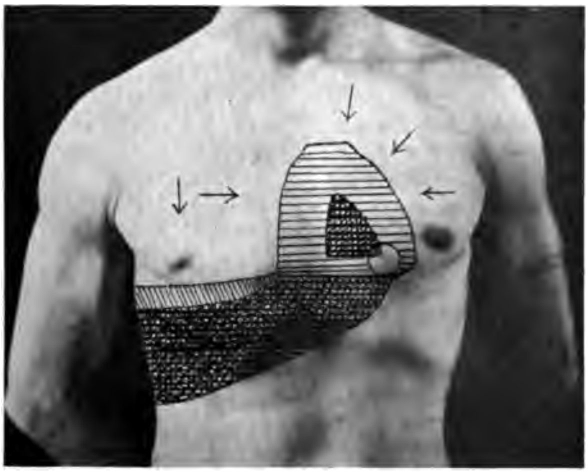
In order to detect spinal deformities the patient should be sitting, muscularly relaxed, and the examiner should note especially: the relative height of the shoulders and of the scapulæ, the depth of the supraclavicular fossæ, and in women the height of the breasts. The spine itself is, of course, also inspected both from a lateral and from an anteroposterior aspect. Small degrees of scoliosis may be emphasized by marking the skin over the spinous processes of the vertebræ with a pencil.

CHAPTER VII

MAL VARIATIONS OF THE PULMONARY PERCUSSION SOUNDS

INDIVIDUAL VARIATIONS

ıre is no invariable normal standard. Actual values must be ined largely by the variation of the two sides of the chest, while rmal range must be learned by experience. Percussion sounds ry with: (*a*) the soft parts overlying the lungs; (*b*) the flexibility thorax; (*c*) the size and shape of the lungs, and their state of ı; (*d*) the region percussed.



85.—Showing normal areas of dulness and flatness caused by and outlining the c] position of the heart and the liver. The lower border of the heart cannot be out- ce it overlies the liver and these organs have acoustically, in so far as percussion is ıd, identical qualities. The heavy shading indicates the part of these organs which ıvered by lung tissue and therefore yield a flat note. The arrows indicate the di- n which percussion should proceed. The light shading shows the areas over which monary resonance is replaced by slight dulness owing to the proximity of the under- er and heart. (See Figs. 86, 167.)

REGIONAL VARIATIONS

teriorly.—The clearest pulmonary resonance is encountered below vicles and at the angles of the scapulæ. In women a diminution ınance and an elevation of pitch—owing to the *mammæ*—begins the second interspace. On the left side *cardiac dulness* begins at per border of the third rib, and on the right, the *hepatic dulness*, the fourth interspace. The note on approaching these organs es progressively less resonant until absolute flatness is encountered

—no lung tissue intervening between the organ and the chest wall. Diminished resonance is also encountered in the *splenic region,* but the spleen cannot be accurately outlined by percussion (Figs. 61, 87). There is less resonance at the sterno-clavicular, than at the sterno-acromial angle, and less resonance over the second rib than over the second interspace. Over the upper *sternum* the osteal quality predominates, below the second rib more pulmonary resonance may be elicited, especially on light percussion.

Shortly before the lower costal margin is reached the note becomes slightly tympanitic owing to the underlying air-containing viscera—

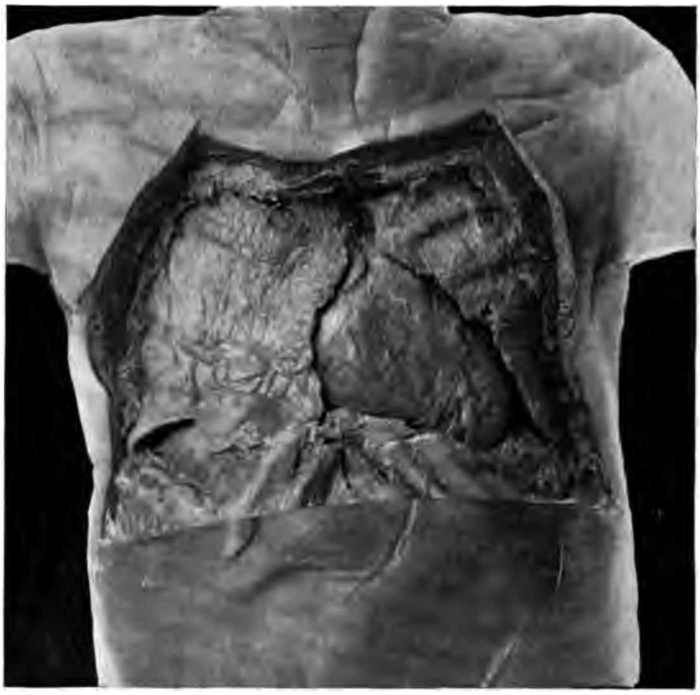


FIG. 86.—Window dissection of the chest from in front, showing the position of the heart and liver which causes the dull areas charted in Fig. 85. The pericardium has been removed. The area of absolute (superficial) cardiac dulness is larger than normal, because the lungs were frozen in the position of expiration. The amount of heart exposed is therefore unusually large. Not infrequently the anterior pulmonary margins overlap in front, leaving very little of the heart exposed. (See Fig. 85.)

stomach and intestines (Figs. 78, 107). On this account liver dulness, especially to the right of the right para-sternal line, often does not extend to quite the lower margin of the ribs unless the organ is enlarged.

On the left side near the lower costal margin a tympanitic area—*Traube's semilunar space*—is encountered. The tympany is caused by that area of the stomach which lies between the left lower margin of the lung, the right border of the spleen and the left border of the liver. It disappears when the stomach is filled with food and in cases of pericardial effusion. It is diagnostically of little importance (Fig. 89).

Dulness over the left lateral pulmonary margin may be caused by a full stomach, and tympany in this region is often due to tion. These facts must be borne in mind or diagnosti made (Fig. 90).

Posteriorly.—The supraspinous fossæ despite the t overlying muscles, gives a certain amount of pulmor especially in women and in emaciated individuals (Fi The upper scapular region is less resonant than the l

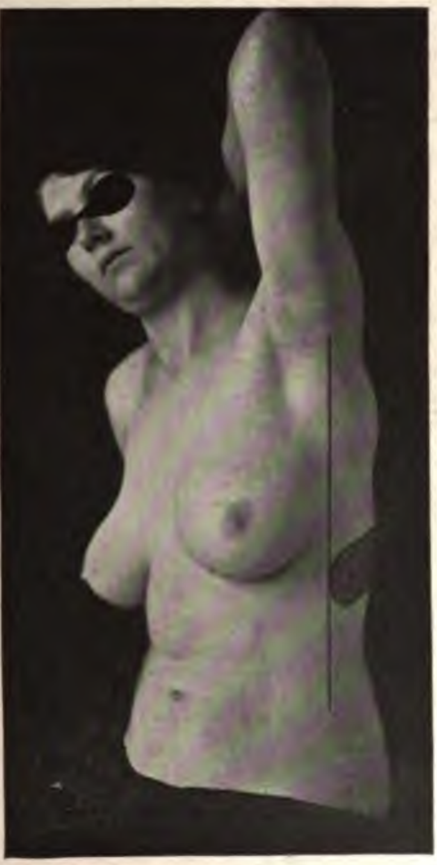


FIG. 87.—Splenic dulness. Percussion of the spleen has but litt organ is enlarged, and when this is the case palpation is a more sat examination. (Compare Figs. 61 and 70.)

over the spine. The interscapular region lies between th two. The infrascapular region gives a clear resonance sion as far downward as the eleventh interspace, but for brings out the dulling due to the liver, as high as the ni eighth rib on the right side. On the left side the note is v by the neighboring influence of the stomach and spleen (I *axillary region* hyperresonance is often encountered; in tions, the stomach imparts a tympanitic, and the liver a d approach the level of the diaphragm.

The percussion outlines of the lung vary with the phase of respiration. One can map out the superior, inferior, and precordial margins. Respiration changes the percussion note, by altering the volume, tension and density of the pulmonary tissue and by displacing organs.

The superior margin of the lung extends 2 to 3 cm. (1¼ inches) above the clavicles at the end of a quiet respiration.

There has been much discussion as to whether, and to what extent the *apices move during respiration.* It seems definitely established that only the anterior and lateral aspects can move and that there is a slight upward movement during inspiration. Over the anterior slope of the apex lies a lid consisting of fibrous fascia, the first rib, the subclavian artery and vein. This lid is hinged from behind. When its anterior part is lifted up during inspiration, it is the anterior part of the apex which feels the movement; the posterior portion lying in front of the neck of the first rib, being nearer the axis, is affected only indirectly (Fig. 93) (Keith, *Journal of Anatomy and Physiology,* May, 1903. Appendix, *Proc. Anat. Soc. Gr. Brit. & Ireland,* May, 1903).

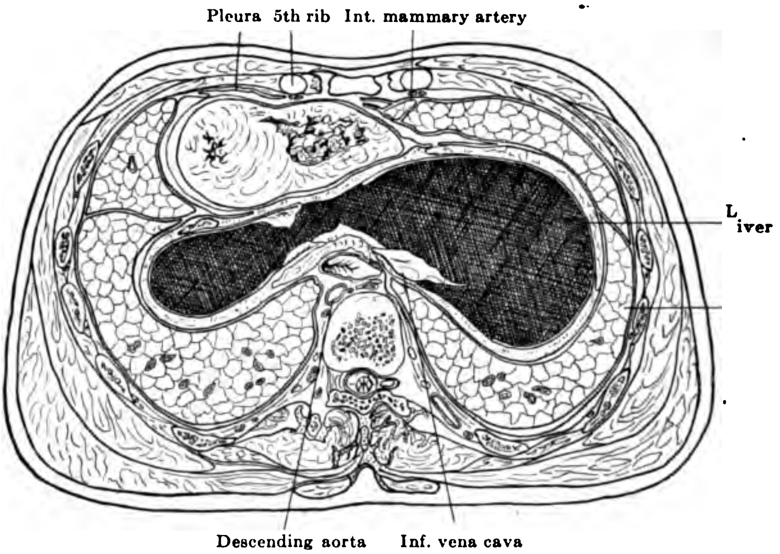


Fig. 88.—Transverse section through the thorax from the lowest part of the ensiform cartilage to the bottom of the ninth dorsal vertebra. The following points are to be noted:

1. Liver dulness is demonstrable by percussion on the right side of the chest only; on the left side this organ is too deeply placed. (Compare Fig. 78.)
2. The liver extends even further to the left than does the heart which rests upon it, only the diaphragm separating these two structures. In case of abdominal distention (ascites, meteorism) the diaphragm encroaches even more upon the pleural cavity than in the normal case here shown. For this reason the pulmonary bases may yield a dull note on percussion in cases of ascites, even in the erect position and in the absence of pulmonary congestion or hydrothorax (upward displacement of the liver).
3. This section passes through the heart 2 cm. above its lowest level, the pericardium which contained half an ounce of serous fluid extended 2 cm. below the level of the heart. In the performance of *paracentesis pericardii* there is less danger of puncturing the pleura if the trochar is introduced in the fifth interspace on the left, close to the sternum. At this point both the pleura and the internal mammary vessels will be avoided. (*Figure redrawn after Braune.*)

Percussion of the Supraclavicular Fossa.—The idea that a failure of the percussion resonance to move upward during forced inspiration indicates apical adhesions or infiltration is erroneous. Such an increase in resonance is not always present under normal conditions, and when so, is

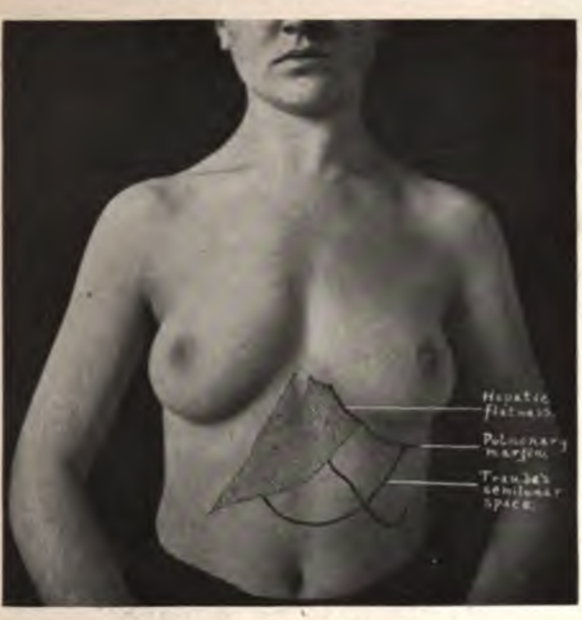


FIG. 89.—Traube's semilunar space.

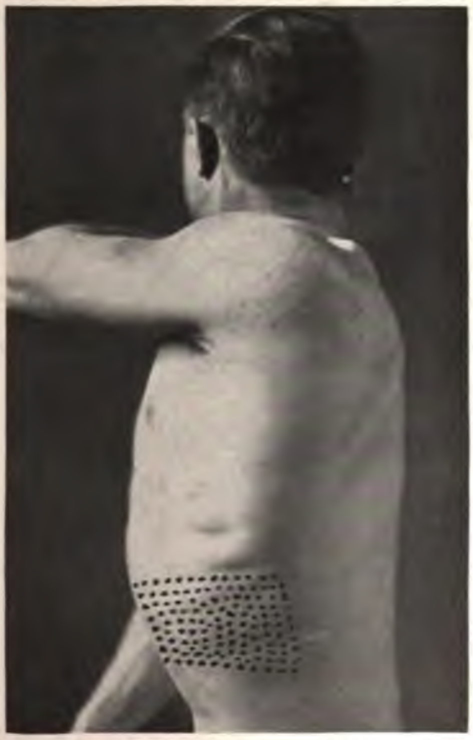


FIG. 90.—Dulness due to a full stomach.

mainly an indication that the apex of the lung has become broader, deeper. As has been stated above, there is practically no upward movement. Since the clavicles move with respiration, they cannot be taken as accurate fixed points, from which to judge of apical expansion.

The Inferior Pulmonary Margin.—During forced, held, inspiration, the percussion note becomes slightly higher-pitched, because of the increased tension of the pulmonary tissue. The lower border of the pulmonary resonance should be noted during quiet breathing and its distance from the level of the seventh cervical vertebra noted. The patient is then asked to take as deep an inspiration as possible and hold it. The line of pulmonary resonance will move downward several centimeters during this procedure. The absence of this phenomenon may be due to pleural adhesions or effusion, pulmonary fibrosis or infiltration (see Fig. 50).

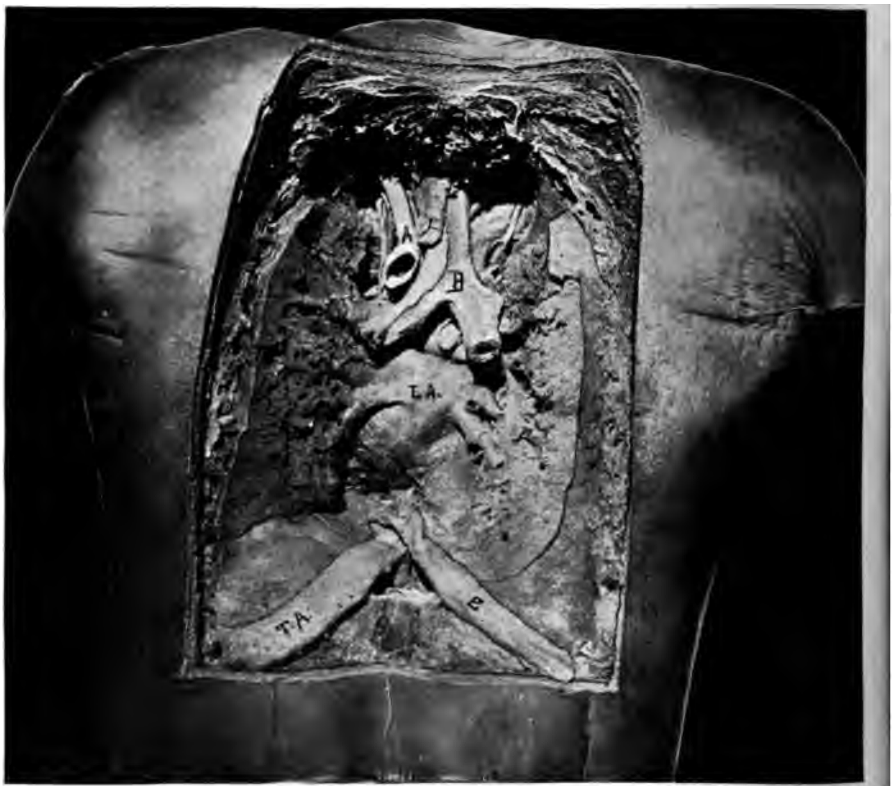


FIG. 91.—Window dissection of the back, showing: 1. Thickness of dorsal muscles through which the percussion vibrations must penetrate before reaching the pulmonary tissue. 2. The bronchial bifurcation over which area the breath sounds are normally broncho-vesicular, the fremitus and vocal resonance intense. *A*, aorta; *B*, bronchial bifurcation; *L.A.*, left auricle; *T.A.*, thoracic aorta; *E*, esophagus. Forcible, two-finger percussion is sometimes necessary to elicit pulmonary resonance at the level of the shoulder girdle. 3. This dissection further illustrates the reason why spinal percussion has practically no value. The distance of the mediastinal viscera from the point percussed and the thickness of the spinal column render the localization of percussion vibrations well-nigh impossible.

During quiet respiration the margins of the lungs move but little. In the *dorsal position*, the anterior margin moves downward about 2 cm. (¾ inch) lower than in the erect posture. In *lateral decubitus* the edge of the lung on the upper side moves

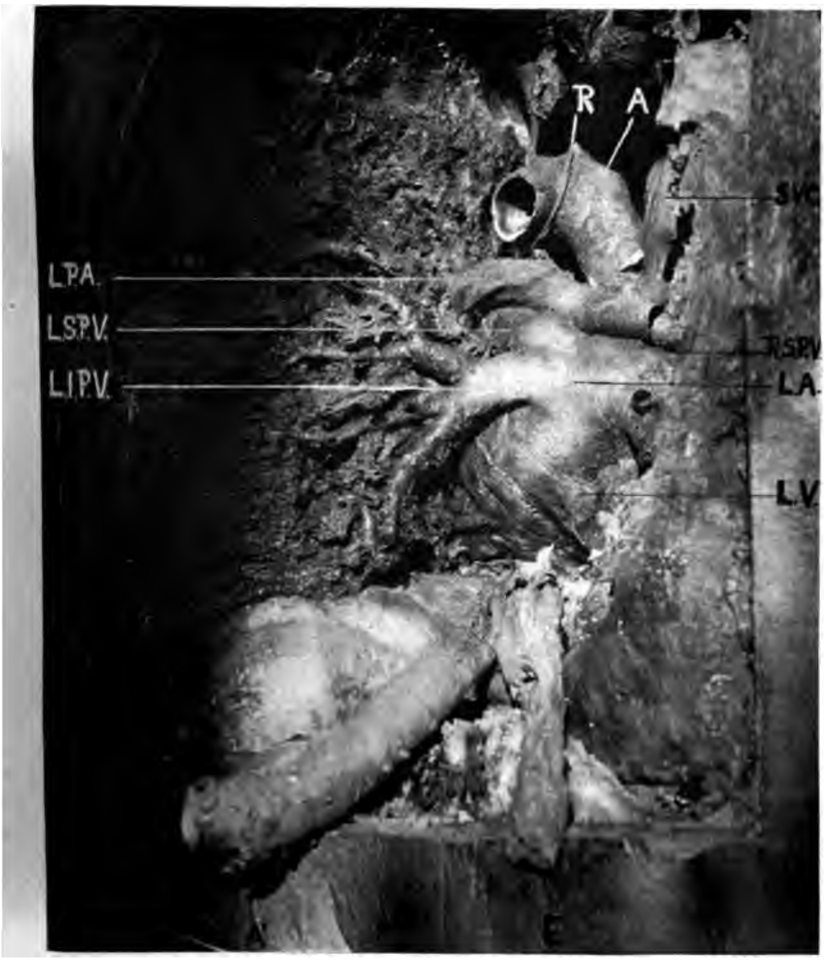


Fig. 92.—Oblique view of the mediastinal viscera from the right posteriorly, showing A, aortic arch; *A′*, thoracic aorta (reflected); *R*, recurrent laryngeal nerve; *L.A.*, left auricle; *L.V.*, left ventricle; *L.P.A.*, left pulmonary artery; *L.S.V.P.*, left superior pulmonary vein; *L.I.P.V.*, left inferior pulmonary vein; *E*, esophagus (reflected). The left recurrent laryngeal nerve is not infrequently compressed in aneurisms of the arch of the aorta. This results in a localized neuritis which is clinically manifested by a husky voice, a "brassy" cough and sometimes "rattling in the throat." The nerve may be compressed indirectly in some cases of mitral stenosis.* The right auricle being greatly dilated presses the left pulmonary artery upward, squeezing the nerve between this vessel and the aortic arch.

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* Fetterolf, G. and Norris, G. W.: "The Anatomic Explanation of the Paralysis of the Left Re urrent Laryngeal Nerve Found in Certain Cases of Mitral Stenosis." *Am. Jour. Med. Sc.*, May 1911

down 3 to 4 cm. During forced breathing the excursion may amount to 9 cm. (3½ inches).

Respiratory displacement of the lung—which occurs through a filling of the complemental space—is most marked in the axillary line. The lower pulmonary border never descends nearer to the lower costal margin than 7 cm. (2¾ inches) on the right, and 5 cm. (1.96 inches) on the left side (see Figs. 9, 10, 11).

Pathologic Displacement.—*Downward* displacement of the lower lung border may be permanent (emphysema) or temporary (asthma). *Upward* displacement of the lower pulmonary border occurs in cases of pleural or pulmonary contraction (fibrosis), and in intraperitoneal enlargement.

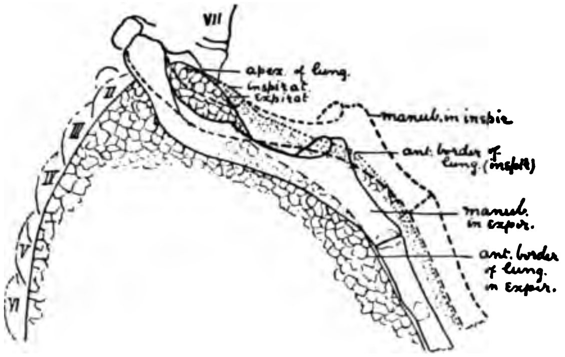

FIG. 93.—A figure to show the relationship of the first rib and manubrium sterni to the apex of the lung (1) in expiration, (2) in inspiration. (*Keith.*) (See p. 97.)

OTHER VARIATIONS

Sex.—In women the upper chest is more resonant as compared to the lower, than in men, due to the habit of upper thoracic breathing. The mammary region is often difficult to percuss on account of the thickness of the tissues.

Age.—In old age the costal cartilages become ossified, resulting sometimes in a higher-pitched, less resonant note. In other cases a more tympanitic quality is heard. These alterations are in part due to thoracic rigidity, and in part to the condition of the lungs, depending upon whether senile pulmonary atrophy, or emphysema has occurred. The rigid thorax tends to prevent deep penetration of the percussion blow.

THE DIAPHRAGM

The position of the diaphragm may be determined by: (*a*) percussing the lower border of the lungs; (*b*) percussing the upper border of the liver and spleen; (*c*) observing Litten's phenomenon; (*d*) Harrison's groove if present; and (*e*) by means of the X-ray.

The lungs do not fill the pleural sac during quiet respiration, but leave a semicircular space—the complimental space of Gerhardt, between the chest wall and the shelving diaphragm. The right phrenic dome is, as a rule, 1 inch higher than the left, but both may be equal or the normal difference even reversed (see Fig. 59). During inspiration a recession downward is noted, but hardly any appreciable flattening, except with forced inspiration. The extent of the excursion in quiet breathing is ¾ inch; in forced respiration 2½ to 5 inches. The diaphragm is normally lowest while sitting, intermediate in standing (active abdominal muscles) and highest in recumbency. In the right lateral decubitus, the right dome is higher (greater weight of the abdominal contents), and *vice versa*, notwithstanding this fact the excursion is greater on the dependent side (muscles act more forcibly against resistance), the upper side remains relatively motionless (decreased excursion because of approximation of the diaphragmatic origin and insertion).

Alterations in arching and mobility are almost a constant factor in pulmonary tuberculosis, especially if there is fibrosis or basal pleural adhesions. In unilateral cases, the fluoroscope, generally shows a high, well-arched dome on the affected side which moves only slightly, and a low flattened dome on the sound side (compensatory emphysema).

SOME PHYSIOLOGIC CONSIDERATIONS

The diaphragm is the chief means of *inspiration.* In quiet breathing the main function of the intercostals is to fix the ribs, and maintain expansion during the descent of the diaphragm. When the latter is hindered or inspiration becomes labored the intercostals act more strongly, so as to raise the ribs and dilate the thorax. In very forcible inspiration other muscles act also: sternomastoid, scaleni, omohyoids, upper part of the trapezii. The scaleni simply supply an anchorage for the first ribs. The rotation of the ribs, from the second, downward is accomplished solely by the intercostal muscles (Hoover). These muscles hypertrophy from overuse—emphysema.

Elevation of the ribs is also in part produced by the diaphragm (normal position is one of arched tension) which acts by contracting over the liver and the abdominal viscera as a fulcrum. The latter are pressed upward by the abdominal muscles. If "the ribs remain fixed the diaphragm cannot be pushed upward, but if the abdominal muscles pull the ribs inward, then the tension of the diaphragm is relaxed and the abdominal viscera under the pressure of the abdominal muscles, can drive it upward and expel the supplemented air" (Hutchison). The diaphragm and intercostals are antagonists although they concur to produce the same result. The liver and the intercostal muscles prevent the diaphragm from drawing the chest in.

When the line of traction and the plane of the diaphragm coincide—when it is neither arched upward nor downward—the diaphragm overpowers the intercostal muscles and the costal margin is drawn toward the median line. But if the diaphragm is arched, either upward or downward, the intercostal muscles gain the upper hand and the rib margin moves away from the median line during inspiration. For the preponderance of either the intercostals or the diaphragm depends not upon the height of the latter but upon its curve and line of traction.

"The direction of movement and comparative movements of the median and lateral portions of the costal margins on the two sides give us valuable information on activation of the diaphragm and the position of its median and lateral parts. The direction in which any portion of the costal margin may move is determined by the resultant of two forces. One is the expression of the action of the intercostal muscles, which always widens the subcostal angle and spreads the hypochondria; the other force originates in activation of the diaphragm and always draws the costal margin toward the median line. When the resultant of these two forces causes narrowing of the subcostal angle, the subcardial or median portion of the diaphragm is less convex than normal. When the entire costal margin is drawn toward the median line, the entire phrenic leaf has lost a large part of its convexity. The movement of the costal margins has nothing to do with excursion of the diaphragm; it is merely the resultant of activation of the intercostal muscles and of the diaphragm. The only subphrenic sign of phrenic excursion is protrusion of the epigastrium and lateral portions of the abdomen."

In order to determine whether the costal border remains stationary or diverges from, or is drawn toward the median line, Hoover gives the following directions: The examiner should place his thumbs symmetrically along the costal borders to serve as indicators as inspection alone may lead to confusion between elevation of the thoracic cage and widening of the subcostal angle. In obese individuals the movements of the costal borders are usually obscured and can be detected only by palpation.

"The significance of the slight arch of the anterolateral portions of the diaphragm is illustrated very nicely in cases of acute cardiac dilatation due to myocardial incompetence from arterial sclerosis. It is a common experience to see these patients brought to the hospital with dilatation of both sides of the heart. During this period there will be symmetrical inspiratory narrowing of the subcostal angle, but the lower and lateral portions of the costal margins move away from the median line during inspiration. As the heart recovers from its acute dilatation, the upper and inner half of the right costal margin will resume its outward movement during inspiration, but the left side of the subcostal angle will be restrained in its outward movement or may continue to move toward the median line during inspiration. This is, of course, due to the fact that the left ventricle is permanently enlarged and depresses the left sternocostal portion of the diaphragm. The symmetry and asymmetry of movement of the two sides of the subcostal angle and the lower and outer halves of the costal margins give much valuable information concerning the total volume of the lungs, and form and size of the heart and heart's sac. Furthermore, an exact analysis of the respiratory movement of each part of the thoracic cage is essential to form an adequate estimate of the ventilatory function of the different parts of the lungs. Such an analysis is also necessary to form an exact idea about the factors in supraphrenic and infraphrenic diseases which may modify the curve in any part of the diaphragm."[1]

Forced inspiration exerts its effects mainly upon the upper chest and the true ribs.

Quiet expiration is due not merely to cessation of muscular contraction—a passive act, the result of elastic recoil—but also to *active* relaxation (of the inspiratory muscles). The muscular effort required by an inspiration is equivalent to raising 100 pounds; a forced inspiration to raising 300 pounds (Hutchison).

Forced expiration is due to the activity of the abdominal muscles, the latissimi dorsi, and lower part of the trapezii. It exerts its effect mainly upon the lower chest and false ribs.

Paralysis of the diaphragm causes bilateral enlargement of the chest, inspiratory widening of the subcostal angle and flaring of the hypochondria. Paralysis of the intercostal muscles produces bilateral diminution in size with subcostal narrowing.

Respiratory pressure changes are most marked in the lower part of the pleural sac, and least marked between the tracheal bifurcation and the heart (Meltzer and Auer).

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[1] HOOVER, C. F.: "Diagnostic Signs from the Scaleni, Intercostal Muscles and the Diaphragm in Lung Ventilation." *Arch. Int. Med.*, Nov, 1917, xx, 701.

During *inspiration* the *interspaces* are pushed inward by the atmospheric pressure. This is most evident in the lower interspaces.

The visceral pleura is retained in contact with the parietal pleura by an "atmospheric ligament" (Keith) due to the atmospheric pressure (15 pounds to the square inch), by way of the bronchii exerting its force against the negative intrathoracic pressure. "The weight required to separate the visceral from the parietal pleura over both lungs amounts to about a ton" (Hutchison), a far greater force than can be brought to bear by the respiratory muscles, hence the two pleural membranes remain in contact. But if one lung collapses owing to a pneumothorax or a pleural effusion, it is dragged toward the opposite side by its fellow even before the presence of an exudate causes displacement by means of actual pressure. The negative pressure exerted by the lungs upon the pleura favors exudation (hydrothorax) when the vascular engorgement of the pleura exists. Very considerable exudation must occur before the pressure becomes positive. This fact is a strong argument in favor of the early aspiration of pleural effusions. The greater the elasticity or tonus of the lung, the greater will be its traction when the opposing lung is collapsed. Hence pneumothorax occurring with relatively healthy lungs, free from pleural adhesions, will cause greater mediastinal displacement and shock, than if the lungs have partially lost their elasticity or are bound fast by adhesions (Hutchison) (see p. 625).

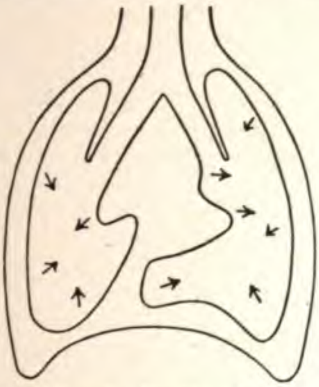

FIG. 94.—Diagram illustrating the direction of the "pull" of the lungs which is due to their elasticity and which occurs when they are relaxed as in pneumothorax. (*After Hutchison.*) Compare Figs. 9, 10 and 11.

The negative intrathoracic pressure is about as follows:

	Mg. Hg.
Normal inspiration	− 10
Normal expiration	− 7
Deep inspiration	− 40
Deep expiration	− 0
Deep inspiration with air passages closed	−100
Deep expiration with air passages closed	+100

CHAPTER VIII

AUSCULTATION

Auscultation, the discovery of which we owe to Laennec, is the act of listening for sounds produced within the body, chiefly those of the heart and the lung.

I. Immediate or direct auscultation is performed by placing the ear against the chest wall. This method is still much used for listening to the sounds produced in the lung. By its employment we get more bone, than air conduction. The sounds heard are generally less loud and clear than those heard through the stethoscope, and accurate localization is not as readily possible. If, and this should but rarely happen, the examination must be made through garments, the unaided ear is preferable to the stethoscope. Vocal resonance may sometimes be better appreciated by the immediate method.

II. Mediate or indirect auscultation is practised by interposing some sound conducting material—a stethoscope—between the ear and the body. This method is now generally employed, especially in ausculting the heart. It has the following *advantages.* The examiner can localize sound more sharply, he can assume a more comfortable position, he comes into less intimate contact with the patient, he can auscult certain regions such as the supraclavicular fossæ, and the axillary apices which would otherwise remain inaccessible.

THE METHOD

It is essential that both patient and examiner be in as comfortable a position as possible. The former will then relax his muscles and breathe more regularly, the latter will be better able to concentrate his entire attention upon what he hears. The sitting posture should be chosen when possible.

For a satisfactory examination it is essential that the part to be ausculted be bared to the skin. After having been put both mentally and physically at ease, the patient should be told to breathe a trifle more deeply than is normally the case, with the mouth open. All raucous, throaty sounds or voluntary expiration must be avoided since they vitiate the results of the examination. It is a waste of time to attempt auscultation of the lungs until the patient breathes properly. A beginning may be made at the top or bottom of the chest and the stethoscope gradually moved from one interspace to another until the entire chest has been ausculted; at each step comparing the sounds of a similar area on the opposite side of the chest, in the anterior, lateral and posterior regions. The supraclavicular fossæ and the axillary apices must not be overlooked.

The bell of the stethoscope is placed firmly against the skin so as to exclude external sounds, but too great pressure is to be avoided, since it increases skin tension, diminishes elasticity and deadens sound. Considerable pressure may occasionally be necessary in order to bring the bell nearer to the source of the sound, as in the case of ausculting the fetal heart sounds, also at times to elicit pleural or pericardial friction sounds by bringing the inflamed serous surfaces into more intimate contact.

Auscultation is sometimes rendered difficult by the presence of cutaneous hair, edema or emphysema. In the first instance the crackling of the hair may be obviated by shaving or by simply wetting the hair. The loud crackling sounds produced by subcutaneous emphysema render auscultation of the intra-thoracic organs quite unreliable if not absolutely impossible.

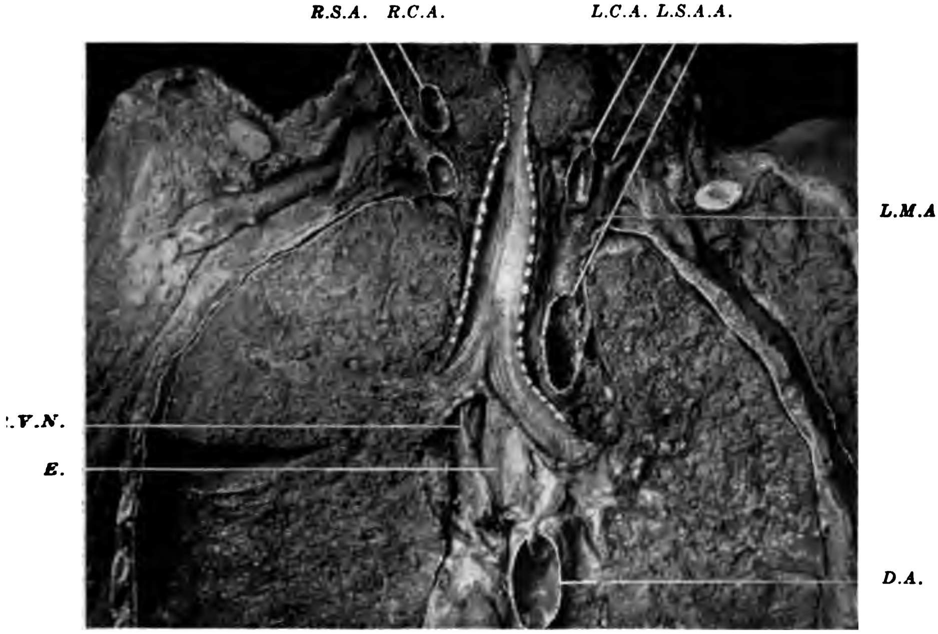

FIG. 95.—Broncho-vesicular breath sounds are often due to stenosis of the upper air passages. If the patient's neck is sharply turned toward either side, if he has hypertrophied tonsils, adenoids, etc., such sounds may be produced in normal lungs. They are to be differentiated from pathologic sounds by the fact that: 1. The harshness is bilateral, is often audible at a distance when the patient breathes deeply. 2. They may be greatly modified by opening the mouth. 3. They are unassociated with other physical signs indicative of pulmonary consolidation.

The illustration shows a stenotic trachea due to enlargement of the thyroid gland. Also the intimate contact of the right pulmonary apex with the trachea, in contrast to the marked separation of these structures on the left side. *A*, aorta; *D.A.*, descending aorta; *R.S.A.*, right subclavian artery; *R.C.A.*, right carotid artery; *L.S.A.*, left subclavian artery) *L.C.A.*, left carotid artery; *L.M.A.*, left mammary artery; *E*, esophagus; *R.V.N.*, right vagus nerve.

Extraneous Sounds, etc.—Practice enables us to concentrate our attention on a particular element of a complex sound and to disregard the rest. Thus when we listen to the heart we ignore the perfectly audible respiratory sounds and *vice versa*. Furthermore, certain other noises must be prevented or equally eliminated from our consciousness, *e.g.*: (*a*) noises in the room; (*b*) sounds produced by friction of the stethoscope against the examiner's finger or patient's skin during the act of breathing or by the rhythmic encroachment of a neighboring rib, or by the fact that some part of the rubber tubing comes into contact with the garments, etc.; (*c*) sounds produced by the interposition of garments between the ear and the chest. Needless to say it is useless to attempt to auscult through stiff materials such as starched linen. (*d*) Contracting muscle produces a sound, hence any muscular tremor or voluntary stiffening of muscles on the part of the patient must be prevented. The examining room must not be cool enough to produce shivering. Muscle sounds are especially common over the pectoralis major in the anterior axillary region and over the trapezius muscle. (*e*) Occasionally the physician may, when the stethoscope has been placed in the auditory meatus, become subjectively conscious of his own circulatory sounds.

In ausculting the chest, sounds are frequently heard which to the beginner, sound like râles. If the patient swallows while one is listening over the chest the sound produced resembles very clearly that produced by a râle. This is especially apt to occur when the patient coughs and then takes a moderately deep breath. As this procedure is employed especially to elicit latent râles, the patient should be cautioned not to swallow. In ausculting the apices care must also be taken not to confuse joint crepitations with râles. The scapulæ, the costo-sternal, sterno-clavicular and shoulder joints may produce such sounds. These fictitious râles can be identified by manipulating the articulations named.

Occasionally when ausculting the axillary regions fine râles are heard during the first moderately deep inspiration. They are of no significance unless they persist during succeeding inspiratory acts.

At this place mention might be made of the *transmission of sounds from the diseased to the healthy side*. Very often the sound produced by large bubbling or resonating râles as well as exaggerated voice sounds and loud cavernous or bronchial breathing on one side may be heard on the opposite side. When this occurs it is always over the upper and posterior portion of the chest near the large bronchi. Mistakes may be avoided by noting the character of the percussion note and the breathing, and especially by tracing the sounds from their point of maximum intensity across to the opposite side. If the sounds are not due to bilateral lesions they will gradually diminish in intensity.

THE INFLUENCE OF POSTURE ON THE PHYSICAL SIGNS

The posture of the patient has certain definite modifying influences upon the results of the physical examination. A sitting position is always preferable. If the patient lies upon the side, percussion of the lower lung yields a slightly impaired note with a tympanitic quality. The former results from the lessened amount of air which the lung contains, the latter from diminution of pulmonary tension and from the resonating property of the mattress. The resonating property of extraneous objects must be borne in mind. For example, pulmonary resonance is greater if the patient is percussed immediately in front of a door (sounding board) then if he stand in the middle of the room. Slight dulness over one pulmonary apex which may be readily demonstrable when the patient sits up, may disappear if percussion is performed in recumbency while the thorax rests upon pillows or a soft mattress (resonators).

In the lateral decubitus the breath sounds in the lowermost lung tend to become muffled and feeble, owing to the decrease of tidal air. In the uppermost lung, on the contrary their intensity may be increased.

THE STETHOSCOPE

The main functions of the *binaural stethoscope* are (1) to prevent lateral radiation of the vibrations with which the air it contains, is charged, thus conducting a larger proportion to the ear, and (2) to exclude extraneous sounds. A large bell furnishes more sound than a small one, because it covers a larger area of vibration-emitting surface. The bell may also act as a resonator and reinforce certain vibrations. Thus a bell 2 inches in diameter enables us to judge accurately regarding intensity and rhythm, but is inferior to a bell of ordinary size or to the unaided ear in the judging of equality and pitch (Flint). Furthermore it is evident that depending upon its shape and the elasticity of the material of which it is made, the resonating quality of the bell must vary considerably. A small bell is also advantageous since it enables us to localize sound more sharply, this being especially important in ausculting the heart and the supraclavicular apices, as well as in children and emaciated individuals in whom the projecting ribs interfere with close application of the bell.

The importance of the thoracic wall in acting as a resonator of the laryngeal vibrations has already been alluded to under vocal fremitus (p. 47). The sounds that we hear are due to both visceral and mural vibrations. The latter sometimes preponderate in intensity and having a lower pitch have a greater volume; whereas the higher range due to the fundamental note is often medically of greater importance. Mural vibrations can to a certain extent be eliminated, and the visceral vibrations studied in isolation by exerting marked pressure, with the bell of a a binaural stethoscope equipped with rubber tubing.

In case of the *mon-aural stethoscope* made with a hollow or solid stem of metal, vulcanite or wood, transmission occurs both through the stem and through its contained air. The thinner the walls of the stem and the bell, the more easily will sympathetic vibrations be set up and transmitted to the ear. A large part of the sound is thus transmitted by the solid stem. This applies to a slight extent also to the modern binaural stethoscope, in which some sound is conducted by the rubber tubing, which tends to reinforce the aerial vibrations. Nor "is it immaterial what thickness of tubing we employ. It must be neither too flexible nor too rigid. High-pitched sounds are conducted along tubes, especially soft tubes much less readily than low-pitched sounds, because of their smaller mass."

In using the binaural stethoscope with rubber tubing, the results of pressure on the thoracic wall are quite different. In case, for instance, that the bell is thick-walled, and possessed of a high fundamental note it will be much less affected by the damped mural vibrations than is the case with the monaural stethoscope.

"A solid body laid upon the chest wall takes up and transmits according to its own elastic properties the vibrations emanating from the latter. When the pressure of the solid body upon the surface is sufficiently increased, the sensible vibrations of the latter are damped, but their energy is, of course, transferred to the body which extinguished them. When, for example, the mon-aural, solid stethoscope is applied to the chest, the sounds heard through it are not deadened by increasing the pressure of contact; on the contrary, they tend to become more intense, and are brought nearer the ear."[1] *The diaphragmatic type* of instrument yields louder sounds than a simple bell, because it covers a large area of sound emitting surface and because the diaphragm prevents the encroachment of the soft tissues upon the lumen of the receiver (Montgomery). Perhaps also the sound waves are amplified by the vibrations of the disc. At all events, this type of instrument should be used as a magnifying lens to study detail and, especially by the beginner, not as the sole method of auscultation. *The sounds heard are not merely intensified, often they are distorted* because the instrument disproportionately magnifies certain sounds.

"Binaural stethoscopes and to a minor degree phonendoscopes are less well adapted for the auscultation of faint, high-pitched murmurs, wheezy sounds and the metallic phenomena."[1]

Pulmonary sounds are often heard better with *the unaided ear,* applied directly to the chest wall, because in such cases we get bone conduction as well as ear conduction. The *early stages of pulmonary consolidation, the tubular quality of bronchial breathing, and at times the diastolic aortic murmur are also better appreciated without a stethoscope.*

On the other hand, *the vesicular element of the respiratory murmur, heart sounds, and generally speaking murmurs, also certain râles are more easily analyzed with a stethoscope.* This is perhaps due to the fact that certain wave lengths bear a definite reinforcing relation to the size of the receiver and the length of the stethoscope tubing.[2]

With a small bell we can localize more sharply but we get less volume. Therefore, wider bells give better results for feeble sounds—weak murmurs, fetal heart sounds, etc. For the latter deep pressure on the abdomen is also requisite.

If a stethoscope with a spring is used, the curvature of the metal parts and ear pieces should correspond to that of the external auditory meatus, since the opening in the ear piece should point directly toward the drum and not toward the cartilaginous meatus. Owing to differences in the angle of individual ears, stethoscopes with metal ear pieces must have adjustable angulations. It is partly for this reason that the author prefers simple rubber tubing without any spring attachment whatever.

The Choice of a Stethoscope.—This is largely a question of personal preference. A small minority still prefer the mon-aural type, perhaps as a matter of habit. Certainly two ears are better than one, especially when one is hampered by extraneous noises. In a choice between many binaural types certain factors must be considered:

1. The ear pieces must fit the external auditory meatus exactly, not only in order to exclude outside noises, but also so that the instrument can be used indefinitely without discomfort or pain. A little time consumed in filing the ear pieces to the proper shape and dimensions is well spent.

2. Instruments of the phonendoscope type—those with diaphragms—should never be used by beginners. They not only magnify, but also distort, sounds. Their habitual use even by more experienced clinicians establishes a false standard of normality, and often renders the examiner more or less helpless in case they are temporarily unavailable.

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[1] SEWALL, HENRY: "The Rôle of the Stethoscope in Physical Diagnosis." *Amer. Jour. Med. Sci.*, February, 1913, p. 234.

[2] CONNER: *N. Y. Med. Jour.*, July 13, 1907.

3. The tubing should not be smaller than the caliber of the metal parts, and should be sufficiently heavy to prevent kinking.

4. An extra bell (receiver) smaller than the usual size is desirable, especially in the practice of pediatrics.

5. The instrument should be light and compact so that it can be readily carried in the pocket.

The author personally prefers the Sansom instrument with two bells, and furnished with an extra diaphragmatic receiver for occasional use.

THE BREATH SOUNDS

The act of breathing causes certain sounds which are known as the breath sounds or the respiratory murmur. The breath sounds may be heard when the ear is applied to an area of the thorax overlying lung tissue. They are composed of: (1) the laryngeal or "bronchial," and (2) the vesicular, elements.

The Laryngeal Element.—During both inspiration and expiration certain sounds are produced in the nose, mouth, glottis, larynx and trachea. They are due to sonorous vibrations caused by a column of air moving through the structures in question (Fig. 96).

If we listen *over the trachea* during the act of respiration we generally hear high-pitched sounds, having a tubular quality, with expiration lasting as long or longer than inspiration; the former having a higher pitch than the latter and being separated from it by a distinct interval. When we listen *over the lung* these sounds have undergone a great modification. The sounds are softer, lower in pitch, expiration is very short, and faint, indeed often it is inaudible, while the pause separating inspiration has practically disappeared.

Stenosis of any portion of the *upper respiratory tract*, such as by nasal or faucial adenoids, markedly increases the intensity of the laryngeal element of the respiratory murmur. Such obstruction may cause broncho-vesicular breathing over the pulmonary apices, thus simulating infiltration of the lung. It is to be distinguished from the latter by its bilateral character, disappearance when the obstruction is removed and by the absence of percussion dulness (Fig. 95).

The primary sound producing vibration in the respiratory system arises in the vibrations of tissues, not of the air. The moving air sets the tissues in motion; this produces a sound, which is in turn conveyed or conducted by the air, as well as by the tissues which surround them—the bronchi and pulmonary tissue.

The relationship is that of the bow, to the violin string, the former being represented by the air current, the latter by the tissues. Furthermore, the rapidity of the current affects the intensity of the sounds (amplitude of the vibrations). When the air current is rapid, more intense breath sounds are produced. Just as the voice sounds produced in the larynx are carried downward into and through the pulmonary tissues, where they are heard as "vocal resonance," so are the breath sounds, which arise at the same source and are spoken of as the "vesicular murmur."

"The chief effect of the bronchial walls is to prevent diffusion, thus allowing the good conducting properties of the air to operate at advantage."

The voice and breath sounds lose much of their intensity as we hear them over the chest wall, (1) as a result of *diffusion*, although this loss is more or less offset by the deep penetration of the bronchi into the pulmonary tissue; (2) owing to *reflection* which occurs when the air-borne vibrations pass through the walls of the bronchi; (3) because in passing from the bronchial walls into the pulmonary septa more wave energy is lost by reflection; (4) as similar loss occurs in passing through the air chambers of a normally distended lung; (5) since they meet with reflection again in passing from the lung to the chest wall, because here

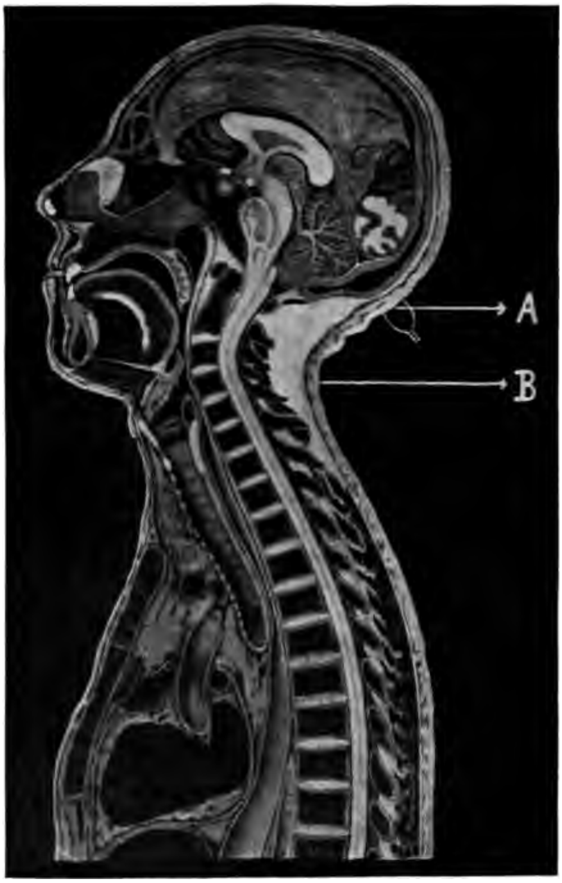

FIG. 96.—This antero-posterior section through the head and thorax depicts the structures concerned in the production and modification of the "laryngeal" element of the breath sounds.

Auscultation over the area "A" during nasal breathing, the mouth being closed, yields pure cavernous breathing (low-pitched sounds with a hollow or reverberating quality, and expiration longer than inspiration). The outgoing air strikes the nares more directly than when the mouth is open; the vocal cords are more closely approximated during expiration and vibrate considerably. The nares and the mouth furnish the cavity for the reverberation of sound, and the occipital bone is a good conductor.

Auscultation over the area "B." If the mouth is open, the air passes directly out of it, the nasal resonator has been eliminated and the cavernous quality as well as the intensity of the breath sounds almost entirely disappear. The results of tracheal auscultation are also greatly modified by oral and nasal breathing. (*Barach.*)

again a distinct "break" occurs in passing from a very light to a fairly heavy medium, as well as (6) diffusion in passing through the chest wall.

The breath sounds as heard over the chest become altered in the character they possess at their source of origin, in regard to intensity, quality and pitch. The *change in intensity* results mainly from diffusion and reflection although absorption, resonance, and perhaps interference also plays a part. "*Changes in pitch and quality*, although explicable to some extent in the elimination of the weaker vibrations as the whole sound becomes more feeble, appear to be due chiefly to the factors of resonance and interference affecting certain vibrations, while exerting little or no influence on other vibrations which go to form the complex group of vibrations which we ordinarily term a single sound" (Montgomery) (see p. 60).

The Vesicular Element.—In addition to the laryngeal sound just described, a vesicular element is added which, when auscultation is practised over the chest, furnishes most of the *inspiratory* pulmonary breath sound. It is due to the separation and distention of the alveoli by the inrushing current of air. The *expiratory* portion appears to arise in the upper respiratory tract. The fact that the sound heard over the chest wall is more muffled, weaker and lower in pitch than that audible experimentally over the actual periphery of the lung is due to loss of vibratory intensity in passing from the lung to the chest wall and from the chest wall to the stethoscope (vibratory reflection).

The Basis for the Assumption of Alveolar Sound Production.—The cubic capacity of an alveolus is 0.00494 mm. (Zuntz).

In long-necked animals, such as the ruminants, the respiratory murmur is much less loud than in the short-necked ones, such as the carnivora.

The estimated total of the alveoli is 400 million. As the average lung volume is 4000 cm., 1 cm. would include 100,000 alveoli. If we assume that in ausculting we hear sound from a depth of 3 cm., we would perceive sound yielded by half a sphere 14 cm. in circumference. In other words, we would hear the combined sound of 1,400,000 alveoli. Since a normal deep inspiration takes 2 seconds, we should hear the sound of 700,000 alveoli per second. Now we cannot recognize as separate sounds, vibrations occurring with a rapidity of 700 per second, as would be the case if 1000 alveoli gave forth sound simultaneously. But alveolar distention is gradual and when a sufficient number of sequent groups produce sound, it is extremely plausible to assume that an audible sound is thus engendered.

Only on such a hypothesis can we assume that sound is produced in the alveoli. The dimensions of an alveolus are so small that there is no possibility of sound production after the manner in which it occurs in the trachea or bronchi. "There can be no question of inspiratory vibration, reflexion, interference, or the formation of horizontal waves where there is no reflective wall because the relationship between the size of the waves and the chambers is too disproportionate" (Geigel, p. 172).

"The total alveolar surface of the lung amounts to no less than 90 square meters, or 100 times the body surface."[1] The interlobar bronchioles end in narrow alveolar ducts and these "expand in turn into comparatively wide infundibuli lined by air cells. This sudden widening out of the air passage is believed to aid in the production of the inspiratory sound heard in ausculting the lungs, eddies being set up in the passage of the air from the narrower to the wider cavity" (Hutchison).

Sahli found in a case of pulmonary hernia due to a sternal fissure that characteristic vesicular breathing could be heard over the hernia during the performance of the Valsalva experiment, in which of course all laryngeal and glottic sounds were eliminated, and proving that at least a part of the vesicular sound originated in the vesicles themselves. The vesicular sound is also noted in cases of cardio-pulmonary murmurs, a factor which further corroborates the foregoing statement (Sahli, Correspondenzblatt f. Schweizer Aertze, 1892).

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[1] R. HUTCHISON: "Applied Physiology," 1908.

The greater part of the *expiratory* sound is produced by the passage of air over the *vocal cords* and through the *glottis*. It contains, as well, certain sounds arising in the oral and nasal cavities and some contributed by the trachea and the bronchi.

The greater part of the *inspiratory* portion arises in the *vesicles*. In as much, therefore, as both, but especially the expiratory portion, have a composite genesis it is not surprising to find that the breath sounds heard over the chest may vary in different individuals, and even in the same person under varying conditions, in regard to intensity, quality, duration and pitch.

CHAPTER IX

NORMAL AND ABNORMAL BREATH SOUNDS

THE NORMAL VESICULAR SOUND

The sound normally heard over pulmonary tissue during the act of breathing—the vesicular murmur—is *regular in rhythm, low in pitch, and soft, breezy or rustling in character.* It is heard throughout respiration, but progressively diminishes in intensity during expiration, the end of which is inaudible. There is only a very brief pause between in- and expiration. It is to be noted that although the duration of these two physiologic phases of respiration is as 5 to 6, the audible duration is as 5 to 1, respectively. In other words *the expiratory sound is much shorter than that of inspiration,* and sometimes it is barely audible. Being a purely passive act, which results from the elastic recoil of the distended lung tissue, it is less harsh, and lower in pitch, as well as shorter than inspiration. The elastic recoil is greatest at the end of inspiration, hence the beginning of expiration is more intense than its end. Furthermore, the glottis is more widely opened during inspiration and the air leaves the chest less rapidly than it enters. The vesicular sound may be imitated by breathing deeply with the lips set in the position which produces a soft "F." In ausculting the lungs, the beginner especially, should *note the character of the expiration,* for it is in this phase particularly that pathologic abnormalities are most apt to occur.

ABNORMAL BREATH SOUNDS

The vesicular murmur may become abnormal owing: (1) to a **change in its character** (intensity, quality, duration, pitch or rhythm); or (2) to the introduction of **new or adventitious sounds,** *i.e.,* (*a*) râles; (*b*) frictions; (*c*) succussion splash; (*d*) metallic tinkle.

THE CLASSIFICATION OF ABNORMAL BREATH SOUNDS

Abnormal breath sounds are more or less arbitrarily classified as (1) **exaggerated,** (2) **broncho-vesicular,** (3) **bronchial,** (4) **cavernous,** and (5) **amphoric.**

Changes in the Intensity and Quality of the Breath Sounds.—**Exaggerated Breathing.**—(*a*) A mere *increase in the intensity* of the respiratory murmur is known as *exaggerated breath sounds,* and is due to increased vesicular activity—more air entering in a given unit of time than is normally the case. Such a condition is *normal in children,* hence the name *puerile breathing.* It may occur in any individual who is breathing hard, as for instance after *physical exertion.* In women the respiratory sounds are louder than in men, especially in the upper, anterior thoracic region, and during expiration (costal breathing). In exaggerated breathing both in- and expiration are harsh and prolonged because the glottis sounds are louder, and on account of the increased pulmonary tension (vicarious "emphysema").

(*b*) **Decreased breath sounds** (feeble, senile, emphysematous, breath sounds) are characterized by the fact that the sounds are faint or short; expiration may be inaudible. This may be due to: (1) *diminished sound conduction*—bronchial obstruction (increased diffusion and reflection)—pleural effusion or thickening (increased reflection) cutaneous adiposity or edema, etc.; or to (2) *diminished sound production*—shallow breathing arising from pain, muscular weakness, thoracic rigidity, pulmonary emphysema.

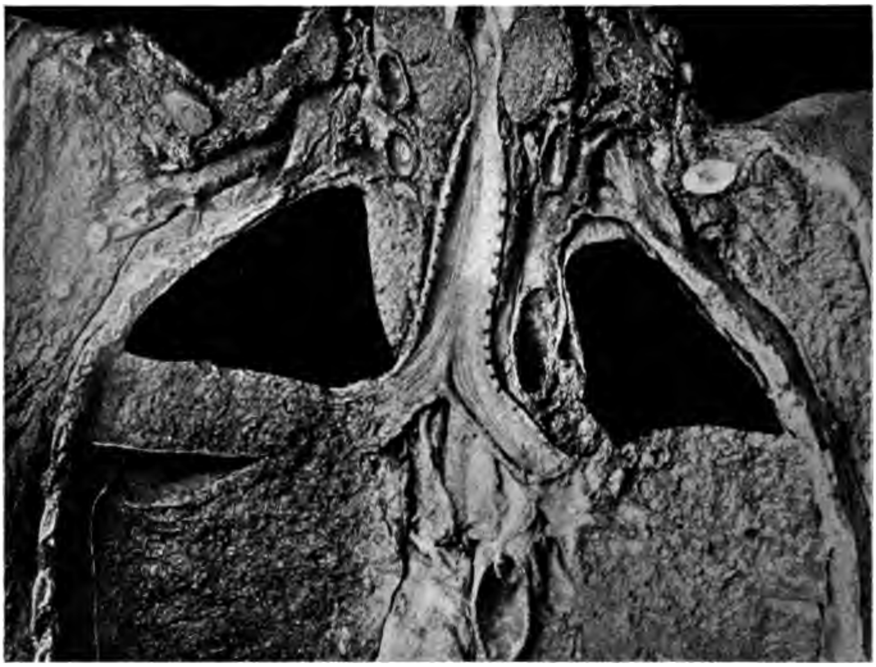


FIG. 97.—Diagram to illustrate the occurrence and non-occurrence of bronchial breathing in pneumonia. On the right side the consolidation spreading inward has reached the bronchus and as a result bronchophony and bronchial breathing are heard.

On the left side there is still a break in the continuity of consolidation between the periphery and the large tubes, and although dulness is marked, bronchial breathing and bronchophony are absent. The breath sounds are feeble or absent. *This partial or entire suppression of the breath sounds, especially of the vesicular element, associated with percussion dulness, and occasional rales on deep breathing, occurs in the early days of a pneumonia much more frequently than bronchial breathing.*

In the early stages of pneumonia and of pulmonary tuberculosis the sounds are often indistinct and muffled.

(*c*) **Breath sounds may be absent** in pleural effusion, closed pneumothoraces with atalectasis, or from occlusion of a bronchus—pressure or exudation. "*Diffusion* of sound between the lung surface, free from adhesions, and the chest wall, shares the honors with *reflection* of sound from the fluid back into the air in the lung, in reducing the intensity of sounds in their passage from lung to chest wall in ordinary cases of pleural effusion" (Montgomery).

Pleural effusions sometimes yield normal or actually increased fremitus, voice and breath sounds even below the level of the fluid. This phenomenon is ascribed by Montgomery to the fact that the *lung is solid* either as the result of exudation, infiltration or compression and being such imparts its vibrations readily to the surrounding fluid without much of a "break" due to reflection. This state of affairs is only inadequately offset by the loss of sound through diffusion in the effusion, so that the end result is at least "normal" if not actually an increased intensity of transmission. A similar effect, although less in degree, may be exerted upon fluid by a collapsed lung. It is furthermore evident that anything which tends to bring the lung near to the chest wall—adhesions, large lungs and small thoraces (in infants)—would further tend to prevent the disappearance of fremitus, resonance and breath sounds, in cases of pleural effusion.

Broncho-vesicular Breathing.—This is a combination of the vesicular and the bronchial types, both the soft vesicular and the harsh bronchial elements being present, either one of which may predominate. Some authorities prefer to use the term vesiculo-bronchial if the vesicular sound is preponderant, and broncho-vesicular when the bronchial sound is the greater. Others prefer to include both these types under one heading, and modify the term by adding such adjectives as slight, marked, harsh, intense, etc.

Physiologic broncho-vesicular breathing is heard over definite locations at which it is normal and can be explained on anatomic grounds. It is considerably influenced by opening, narrowing or closing the mouth or the glottis; and tends to become more intense as a main bronchus is approached. It is normally heard below the right clavicle especially at the second costo-sternal articulation and posteriorly at the tracheal bifurcation. Auscultation over the *manubrium* yields a soft vesicular murmur. Bronchial breathing over this area may occur if the anterior mediastinum is encroached upon by, (1) persistent thymus; (2) substernal thyroid enlargement; (3) edema of the mediastinal tissues; (4) glandular enlargement; (5) dilatation or aneurism of the aorta; (6) tumors. (Warfield.) *The pathologic type of broncho-vesicular breathing* may occur over any portion of the lung and is relatively uninfluenced by alterations in the oral resonator (breathing through the mouth or nose). It occurs when both normal vesicles and infiltrated pulmonary tissue exist together, as in incomplete consolidation. (Figs. 98 and 99.)

Bronchial Breathing.—By this term which is synonymous with tubular breathing, we understand a *loud, harsh, high-pitched, snorting type of breath sound, with a distinct pause between inspiration and expiration, the latter being even longer, more harsh and higher-pitched than the former.* The soft, low-pitched, rustling or breezy vesicular element is entirely absent. The breath-sounds normally heard over the trachea are often used as an example of bronchial breathing, but the tracheal sound is generally more harsh and lower in pitch than that which occurs pathologically over consolidated lung tissue. As in normal breathing, the primary sound *originates in the upper respiratory tract.* Bronchial breathing is characteristic of consolidated lung because conditions are less unfavorable for sound transmission than is the case in normal lungs. The vibrations pass from the bronchial wall to the surrounding consolidated lung and thence to the chest wall without undergoing a

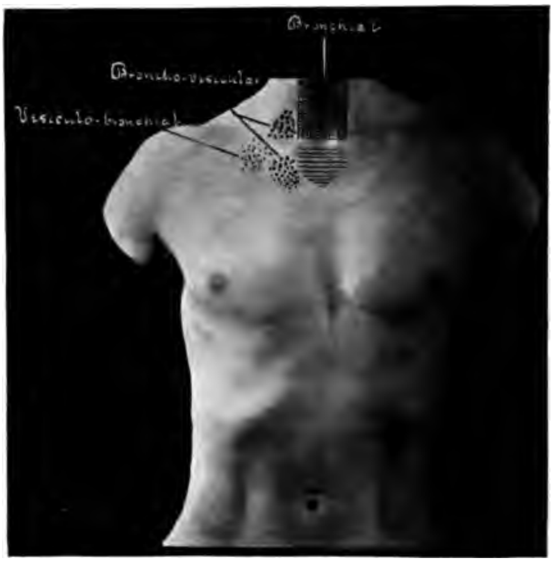


FIG. 98.

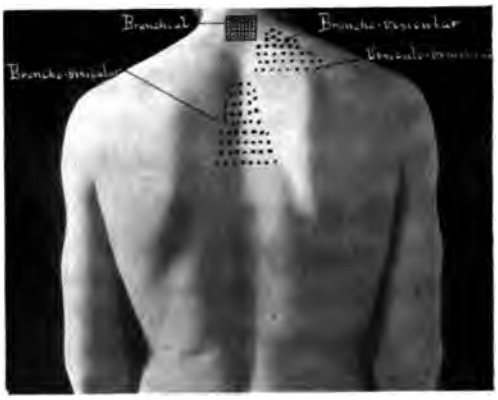


FIG. 99.

FIGS. 98 AND 99.—These figures illustrate the areas over which breath sounds are no mally harsh. The term vesiculo-bronchial as here used signifies vesicular breathing with very slight degree of the bronchial element. In other words, the sound heard is just a trifl more harsh and high pitched than over the corresponding area on the opposite side. Th degree of harshness which is subject to considerable individual variation, is mainly due t the proximity of the right upper lobe to the trachea anteriorly, and posteriorly, in additio to the tracheal bifurcation. (Compare Figs. 49, 52, 75, 95, 104.)

serious "break" in the process of transmission. This is so because bronchus, solid lung and chest wall are acoustically more or less identical and there is, therefore, not much chance for diffusion or reflection of the vibrations at the different tissue junctions.

Montgomery has shown that *lung under normal tension transmits sound much less well than either relaxed or consolidated pulmonary tissue.* "The occurrence of weaker sounds over the normal chest than over the chest where there is consolidation is due not only to favorable conditions on the diseased side, but also to unfavorable transmission facilities on the normal side. These are the main factors in explaining why the side containing the larger amount of air yields weaker sounds at the periphery of the chest than that containing the greater amount of solid tissue."

The term bronchial breathing is sanctioned by usage, but is not descriptive as signifying the sound normally heard over the bronchi.

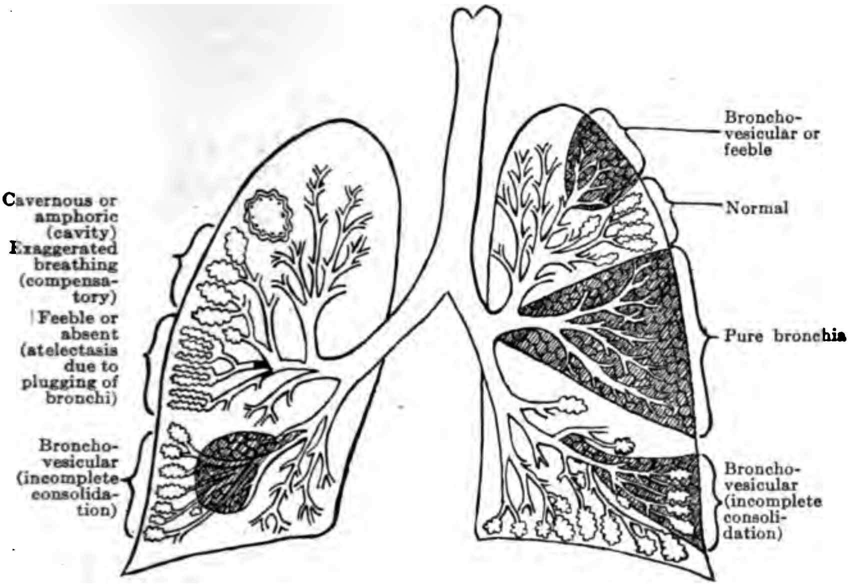

Cavernous or amphoric (cavity)
Exaggerated breathing (compensatory)
Feeble or absent (atelectasis due to plugging of bronchi)
Broncho-vesicular (incomplete consolidation)
Broncho-vesicular or feeble
Normal
Pure bronchia
Broncho-vesicular (incomplete consolidation)

FIG. 100.—Diagram illustrating different types of the breath sounds in various pathologic conditions.

The type of bronchial breathing heard above and sometimes directly over pleural effusions has a similar genesis and is due to associated compression or consolidation of the lung.

Bronchial breathing occurs characteristically over large areas of complete pulmonary consolidation such as in pneumonia. It frequently is not demonstrable until the third or fourth day after the onset. The consolidation begins at the periphery and gradually spreads inward toward the hilus and although dulness occurs quite early, bronchial breathing and bronchophony do not appear until the entire fan-shaped area of lung tissue lying between the pleura and the large bronchi has become consolidated (see Fig. 97).

Bronchial breathing is never heard over normal lung tissue. It may be imitated by a harshly whispered "Ha" or by a guttural "Ch."

Cavernous Breathing.—This type of breathing may be heard over cavities and open pneumothoraces. It is *low in pitch, and has a hollow reverberating quality.* Frequently the expiration is even lower-pitched than the inspiration, the relations of bronchial breathing being thus reversed. As pointed out by Flint, it is more apt to be confused with normal vesicular breathing, than with the bronchial type, and is to be differentiated from the former "only by the absence of the vesicular quality in the inspiratory sound" (Flint). The expiration is always prolonged and puffing (Loomis). Phonetic equivalent = whispered "Who."

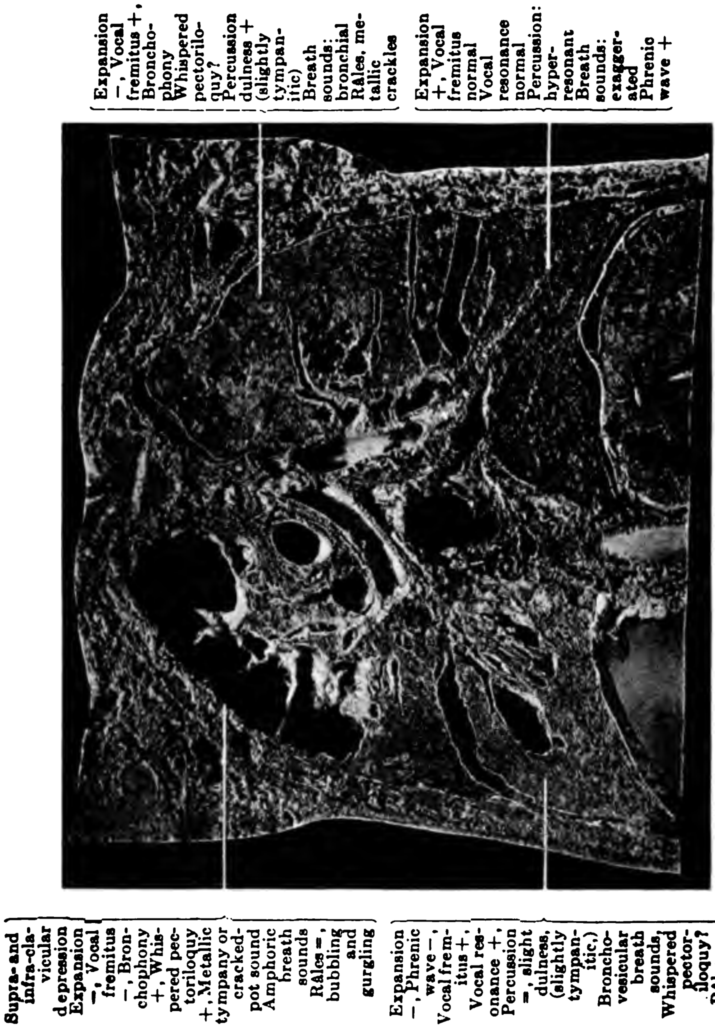

Expansion –, Vocal fremitus +, Broncho-phony Whispered pectorilo-quy? Percussion dulness + (slightly tympan-itic) Breath sounds: bronchial Râles, me-tallic crackles
Expansion +, Vocal fremitus normal Vocal resonance normal Percussion: hyper-resonant Breath sounds: exagger-ated Phrenic wave +
Supra- and infra-cla-vicular depression Expansion –, Vocal fremitus –, Bron-chophony +, Whis-pered pec-toriloquy +, Metallic tympany or cracked-pot sound Amphoric breath sounds Râles =, bubbling and gurgling
Expansion –, Phrenic wave –, Vocal frem-itus +, Vocal res-onance +, Percussion =, slight dulness, (slightly tympan-itic,) Broncho-vesicular breath sounds, Whispered pector-iloquy? Râles = coarse and fine crepi-tant and crackling

FIG. 101.—Pulmonary tuberculosis (*rear view*). The disease is extensive and advanced. The *left upper lobe* has been completely destroyed (cavitation), and is traversed by large unsupported blood-vessels (large hemorrhages likely). The cavity communicates freely with a large bronchus. The *left lower lobe* is infiltrated but contains a cavity and some air-bearing tissue. The *right upper lobe* is completely infiltrated and contains some small deeply situated cavities. The *right lower lobe* is practically free from disease.

Some authors deny the existence of cavernous breathing altogether, classing it as a variety of bronchial breathing—low-pitched bronchial. Cavernous breathing may be imitated by breathing into the hollowed hands It is heard normally over the occiput, especially if the mouth be closed (see Fig. 96).

Like bronchial breathing, cavernous breathing has its origin in the upper respiratory tract. In the former, however, we are dealing not merely with ordinary bronchi but also with excavations which possess resonating properties.

Only rarely is the genesis of this sound due to the fact that the respiratory air current actually enters a cavity and sets up vibrations within it. What generally occurs is that vibrations in the neighborhood of, and external to the cavity, set up sympathetic vibrations within the cavity, which then acts as resonator, reinforcing and modifying the original sound; imparting to it the hollow reverberating quality which we recognize as "cavernous" or the higher-pitched metallic sound which we designate as amphoric breathing.

It is hardly necessary to point out the fact that even large cavities may exist without revealing their presence by cavernous or amphoric breathing. When more or less filled with mucus, pus, or blood, the cavity may be "silent" or only manifested by localized râles.

Amphoric Breathing.—This is a *high-pitched form of cavernous breathing*, with high ringing overtones which impart a *metallic quality*. It may be **heard**: over tense, smooth, stiff-walled cavities, in size not less than a walnut, especially if superficially placed; and over pneumothoraces. It is analogous to the metallic ring of percussion (see p. 70).

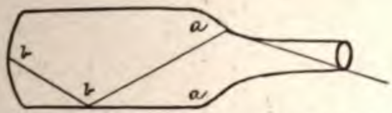


FIG. 102.—Diagram illustrating the production of metallic breath sounds—amphoric breathing—in large stiff-walled cavities.

If we blow into a cylinder such as a test-tube, in such a way as to cause sound waves by reflection, we get a strong fundamental note, with weak harmonious overtones, but if, as with the bottle we add a neck relatively small in proportion to the size of the cylinder, and sufficiently long, we get a peculiar high metallic quality. This is the result of unrhythmic vibrations, due either to the angle of reflection or to the force of the blow, by virtue of which the air in the lee of the neck (see Fig. 102) does not begin to vibrate until after that beyond has been set in vibration. In the former instance we produce cavernous, in the latter amphoric, sounds.

Amphoric sounds may be heard over a closed cavity, or a closed pneumothorax; in these cases the metallic quality is due to a quick, local percussion of their walls by means of the neighboring air columns.

Although amphoric breathing is more intense, and generally of higher pitch than cavernous breathing, the *essential difference lies in the metallic quality of the former*. If we blow softly across the neck of a bottle the sound will be cavernous, if intensely, it becomes amphoric. The pitch depends on the length of the air columns and on the size of the opening. The metallic quality depends on the generation of very unrhythmic vibrations—on the suddenness of the entrance or impact of the vibrations. It is for this reason that the amphoric quality is *often only apparent during forcible breathing, just as a metallic percussion note, and cracked-pot sound can generally only be elicited by quick, forcible percussion.*

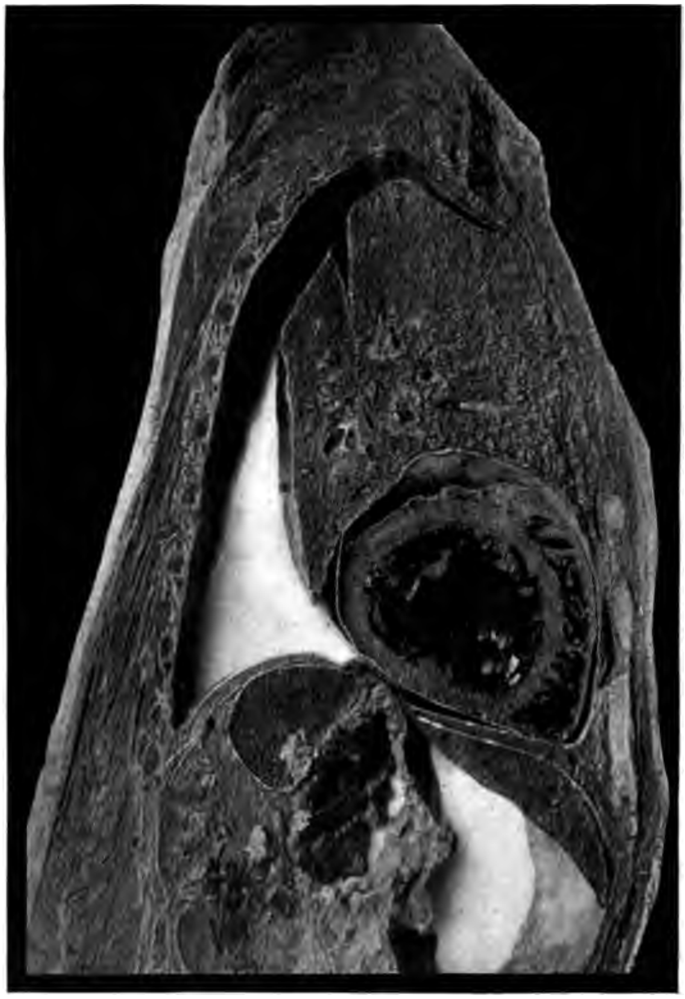


Fig. 103.—Cardiac Hydrothorax. A case of rheumatic mitral and tricuspid disease, with marked cardiac dilatation, auricular fibrillation, etc., under observation for many years. Died in a state of anasarca. Body frozen in the recumbent posture.

The diaphragm is depressed. The effusion reaches to the uppermost limits of the pleural cavity. The lung is compressed, and the dilated heart, displaced downward.

The pericardium contains a small effusion. The following physical signs were noted:

Over the back and the sides of the chest: A flat percussion note, with absent vocal fremitus and resonance and breath sounds. Over the front of the chest: A short, high-pitched, slightly tympanitic note (Skodaic); exaggerated breath sounds, increased vocal fremitus and resonance (compression); subcrepitant râles (congestion and serous exudation); in some areas distant broncho-vesicular breath sounds. In others suppressed, breath sounds (compression of vesicles or of bronchi). In the upper anterior axillary line egophony was elicited.

Metamorphic Breathing.—This term is sometimes applied to different varieties of mixed breath sounds. Thus, breath sounds with a feeble indistinct beginning may end as the pure bronchial type, or the pitch of the bronchial element may change, or inspiration may begin as a broncho-vesicular and end as an amphoric sound. The change is generally attributed to opening of an occluded or occlusion of an open bronchus.

Asthmatic Breathing.—This form of the respiratory murmur is heard during paroxysms of asthma, frequently in emphysematous subjects. Both inspiration and *expiration* are increased in intensity, especially the latter, which is *high-pitched, prolonged and wheezing in character*, and generally associated with musical râles. It is frequently audible at a distance from the patient.

CHANGES IN RESPIRATORY RHYTHM

Cog-wheel Respiration.—In this type of breathing, inspiration instead of producing a soft, continuous sound, as is normally the case, is interrupted, so that it occurs as a series of jerks or puffs and pauses. This occurs chiefly in the upper lobes or at the anterior margins of the lungs. It is due to irregular inflation and expansion of the pulmonary lobules. It may result from pleural adhesions and is a frequent phenomenon in tuberculous cases, but may also occur as a result of asthma, pain or fatigue of the respiratory muscles. (For visible changes in respiratory rhythm see p. 41.) It is sometimes met with in healthy people with apparently normal lungs.

CHAPTER X

ADVENTITIOUS BREATH SOUNDS

RÂLES AND FRICTIONS

Râles are more or less musical sounds which originate in the vibration of exudate in the respiratory passages. The exudate practically acts like a reed in a wind instrument. Genetically all râles are moist, but sometimes they are classified as "moist" and "dry." These are undesirable terms however, and should not be used.

If a râle is described simply as a "dry râle" or a "mucous râle" it connotes nothing definite and further is not along the lines of scientific accuracy. Further, the term "dry" as applied to râles seems paradoxical, for it is almost impossible to conceive of a "râle" that does not, to some extent at least, depend on moisture or increased turgescence for its causation.

Râles vary in quality, size, pitch, time, intensity, duration, distribution, according to the size of the air chambers (bronchi-bronchioles) and the character of the exudate.

Râles are arbitrarily classed in five groups, a classification based on their primary auditory characteristics.

1. **Sonorous râles,** loud, snoring and low-pitched.
2. **Sibilant râles,** whistling, piping, squeaking, hissing, humming. High-pitched.

These two varieties constitute what is known as the musical râles. Their sound is longer and more continuous than that of the other varieties. They are generally caused by a thin, tenacious exudate, and are most characteristically heard in cases of asthma. They are considerably influenced by coughing.

3. **Crepitant râles** are very fine, chiefly inspiratory, constant, clear-cut, high-pitched, uninfluenced by coughing, probably alveolar in origin. They are also known as consonating râles. They are heard in association with bronchial breathing, and seem to originate close to the ear. They may be imitated by rubbing a lock of hair; or by separating the fingers, which have been moistened by saliva. They often have a metallic quality, especially when associated with cavitation, or consolidation. They may be heard in the early and the late stages of pneumonia (crepitus indux and redux). Also in broncho-pneumonia, infarction, pulmonary tuberculosis. They are *the only râles produced in the alveoli and indicate parenchymatous involvement of the lung.*

4. **Subcrepitant (Crackling) Râles.**—These are coarser and lower-pitched than crepitant râles. They may be simulated by rolling a dry cigar between fingers, or sprinkling salt on a hot stove. They originate in the bronchioles, and are heard in cases of bronchitis, pulmonary congestion and edema. Crepitant and crackling râles may be closely simulated by friction sounds.

5. **Bubbling and Gurgling Râles.**—These are also known as mucous liquid râles. They are coarser and lower-pitched than crackling es. They are generally heard over the large bronchi, or cavities, and cases of pulmonary edema. The so-called "death rattle" is due to uid exudation in the trachea, and belongs to this class.

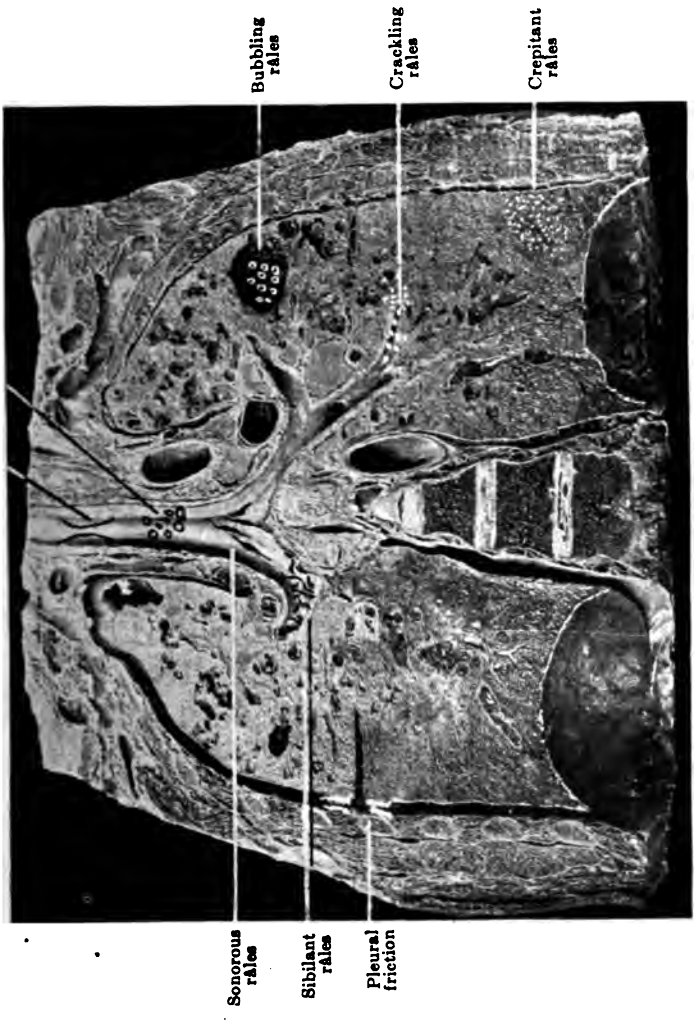
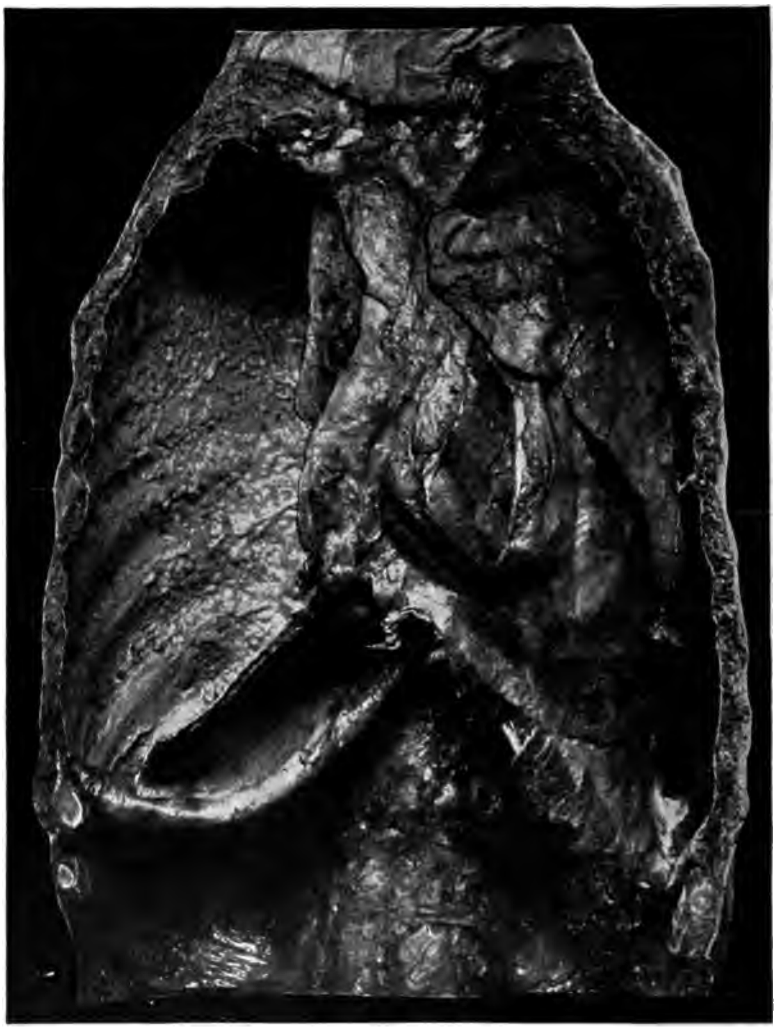


FIG. 04.—THE PRODUCTION OF RÂLES. The site of its production and the character of the exudate determine the acoustic qualities of a râle. This figure diagrammatically illustrates the localities at which different types of râles may be produced. It will be noted that only the crepitant râle is produced in the alveoli, all the other types having their seat of origin in the bronchi, bronchioles, or in cavities.

Bubbling râles are due to fluid—serum, mucus, pus or blood in the nchi. When the fluid is thin, we get a bubbling quality, when thick ertain sharpness or stickiness. The quality depends on the surrounding lung tissue. In consolidations a clear, ringing quality is noted. **bbling râles** vary in size, with the site of production, and with the ntity and character of the exudate.



Expansion —, interspaces full. v. fremitus O. v. resonance O. percussion: flat. Breath sounds O

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FIG. 105.—Pyothorax. Large post-pneumonic empyema. The whole *right pleural cavity* was filled with a thick purulent exudate, some of which may still be seen adherent to the costal pleura. *The right lung* is completely atelectatic, and has been compressed into a small fibrous mass. *The heart* is displaced to the left, and the diaphragm, downward. *The left lung* in its lateral aspect is compressed by the heart. The body was received in the dissecting room from the Anatomical Board, the clinical diagnosis having been "pneumonia."

The **significance of râles** lies mainly in their (*a*) quality and (*b*) distribution, as indicating: (1) the area of activity of the pathologic process; (2) the character and stage of the lesion; (3) the localization—in the lung or pleura.

The primary terms crepitant, crackling, bubbling, sibilant and sonorous, should be further qualified by descriptive adjectives indicating their size, pitch, number, location and association with the phases of respiration, *e.g.*, "numerous, fine, high-pitched, clear-cut, metallic râles heard mainly at the end of inspiration."

Friction Sounds.—These are also spoken of as friction râles. They may be heard over inflamed serous membranes and are due to mechanical attrition of the roughened surfaces. They occur in the pleura, pericardium, and rarely peritoneum. They are characteristically described as dry and leathery (like a sound produced by riding on a new saddle).

They *differ from râles* in that they tend to be (1) localized (in the case of pleuritis to the axilla), (2) unilateral, (3) heard both with in- and expiration. (4) They do not disappear with coughing, (5) they are increased by pressure, (6) they sound close to the ear, (7) they are attended by pain and often tenderness. They may be synchronous with the heart, even if only the pleura is involved.

It is by no means always possible to differentiate between an intrapulmonary râle and a pleuritic friction, but the following points often aid n arriving at a correct conclusion.

RÂLES	FRICTIONS
(*a*) Affected by coughing, or breathing, and vary spontaneously.	(*a*) Constant, not altered by coughing or breathing.
(*b*) Sounds are often "moist."	(*b*) Sounds are more "dry," leathery, creaking.
(*c*) Seem more distant.	(*c*) Seem closer to the ear.
(*d*) Uninfluenced by pressure on the chest wall. Unaccompanied by local pain.	(*d*) Increased by pressure, accompanied by pain, which is also increased by pressure.
(*e*) Distribution more general, more widely diffused. Usually bilateral. Commonly heard posteriorly or near bronchi.	(*e*) Localized in extent, generally unilateral. Most common in axillary region.
(*f*) May be musical.	(*f*) Rarely musical.
(*g*) Occur in showers, are uniform in size.	(*g*) Size and character of component sound elements vary.

Both râles and frictions may be closely simulated by (1) muscle sounds, (2) hair on the chest (see p. 105).

Method of Eliciting Râles.—If râles are suspected to be present but are not heard in the course of ordinary breathing, the patient should be asked to breathe more deeply; or to cough, and afterward breathe deeply. The crepitant râles of early tuberculosis may thus be elicited (**latent râles**). They are also more apt to be heard before or during the period of the morning cough, when such is present. Owing to the faintness of their sound, râles are only rarely heard at a distance from the seat of their origin, although it is said that they are occasionally conducted along the ribs and may even be heard in the opposite lung.

Certain tinkling, gurgling and splashing sounds may be heard over the chest, especially near the spinal column, as the result of liquid passing down the esophagus when the patient swallows. These sounds are often marked and delayed in the presence of a diverticulum.

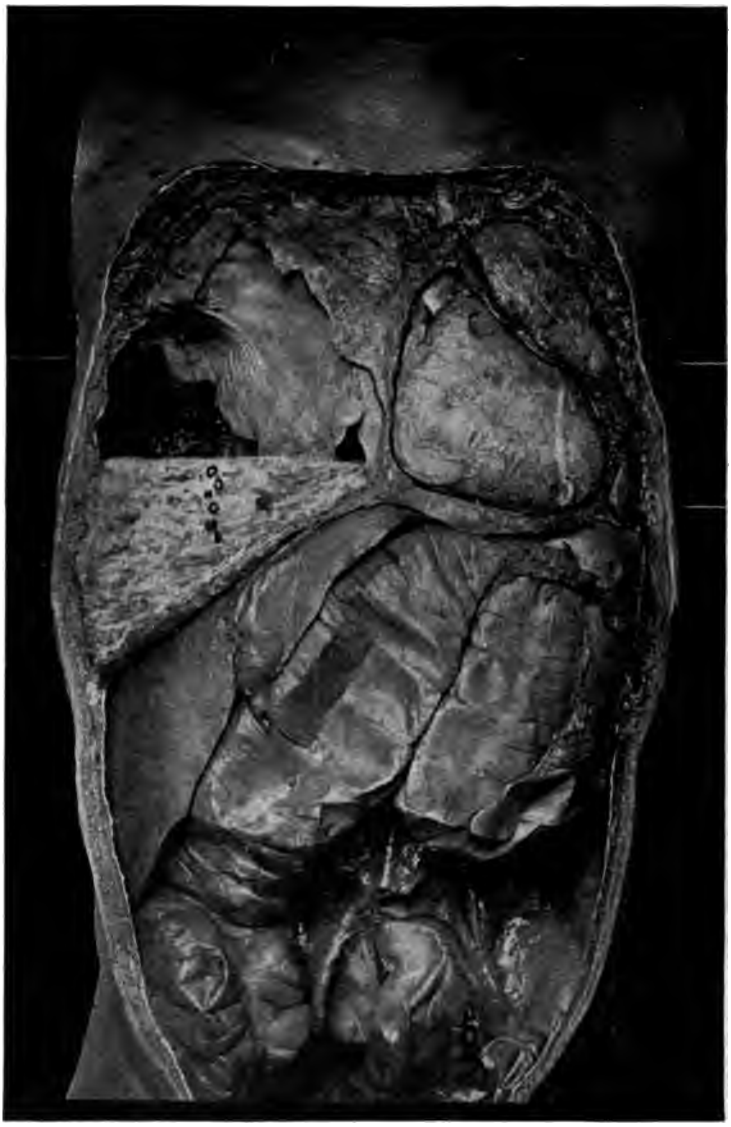
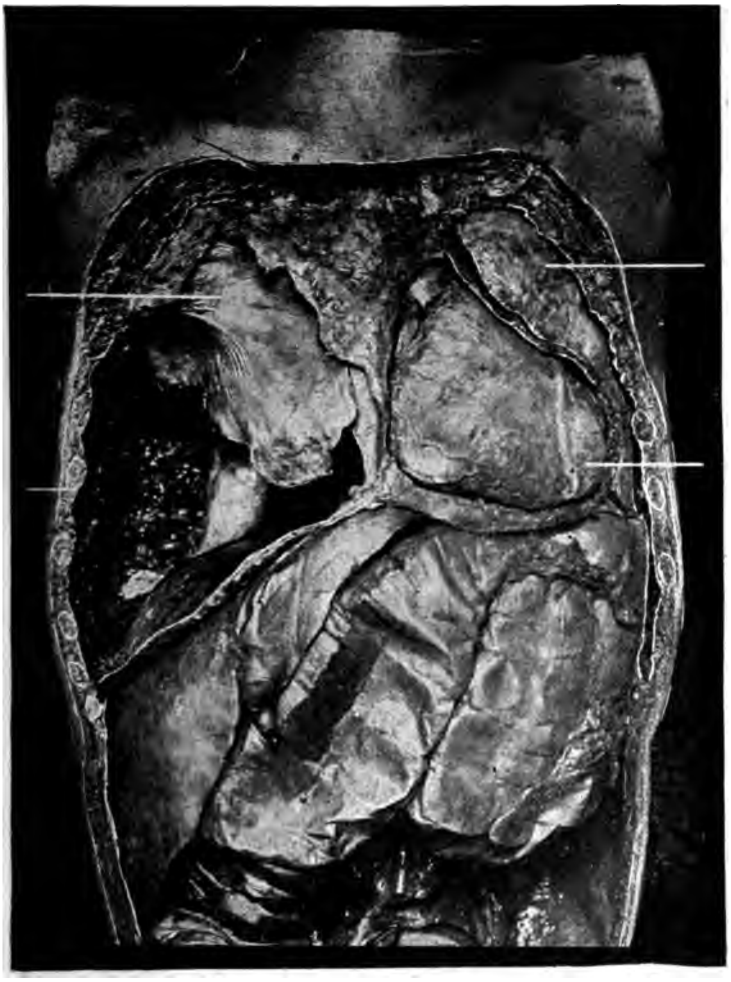

Expansion —. v. fremitus O. v resonance O. tympany +, Breath sounds, O, metallic tinkle +, succusion splash +, bell tympany +
Interspaces full. Expanison —, Phrenic wave O, v. fremitus O. v. resonance O. Movable dulness + +. Breath sounds O, Metallic tinkle +
Expan +. v. f itus + resona +. hy resona breath sounds
Cardia impuls displac down- outwa phreni ward +

FIG. 106.—HYDRO-PNEUMOTHORAX. The right pleural cavity is represented as partly filled with fluid, partly with air. The rupture of the lung has occurred below the present fluid level, so that the fistulous tract opens into the effusion and bubbles are given off during respiration. The right lung is collapsed. The left lung is functionating compensatorily despite its compression. *Succussion splash* may be elicited because *both air and liquid* are present in a large cavity. *Metallic tinkle* is due to bubbles from the fistulous tract passing up through the effusion, *not* to drops of liquid falling upon the surface of the effusion.


Expansion +. Vocal resonance +. Vocal fremitus +, percussion : hyper-resonant. Breath sounds : exagger-ated.
Cardiac impulse and left border displaced to left.

IG. 107.—Large Right-sided Empyema. The *diaphragm* is flattened and depressed, ng the liver downward. The heart is pushed to the left, compressing the left lung, h is contracted and exposes the heart more than is normally the case. The *right lung* is pressed and atelectatic, and in part adherent to the chest wall. The *right pleural* y contained a large quantity of thick purulent exudate, depressing the diaphragm and on that side. On the *left side* the *diaphragm* is pushed upward and flattened against hest wall by the colon. Note how a distended stomach or colon may mechanically rrass the heart. This has an important bearing upon symptomatology and treatment ses of cardiac decompensation and angina pectoris. (*Specimen the property of Dr. . Thomas.*)

SUCCUSSION SPLASH

This may be heard on ausculting over large tense chambers which contain both liquid and air. It is elicited by having the patient cough, suddenly move the body from side to side, or shake his chest.

Succussion splash like the other metallic auscultatory phenomena owes this quality to the suddenness with which the liquid is thrown into motion, unrhythmic vibrations being thus originated. For its production, a stiff-walled cavity of considerable size is necessary. It, therefore, rarely occurs in the chest except in *hydro-pneumothorax*. Pulmonary cavities are generally too small, too flaccid and their contents too viscid, to produce the characteristic sound. It may be simulated by imparting motion to the liquid in an atonic stomach, or by shaking a small quantity of water in a bottle. It can sometimes be heard at quite a distance from the patient, especially if the pneumothorax communicates with a bronchus and the mouth be open. It is often noted in cases of diaphragmatic evisceration or hernia. It is said also to occur over very large pulmonary

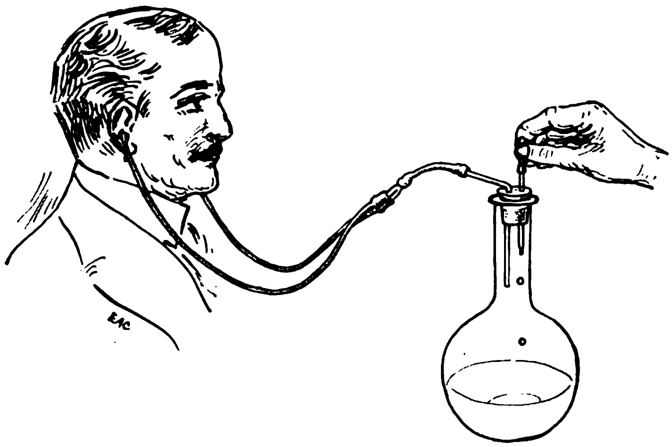


Fig. 108*A*.—The currently accepted but erroneous explanation of the genesis of metallic tinkle.

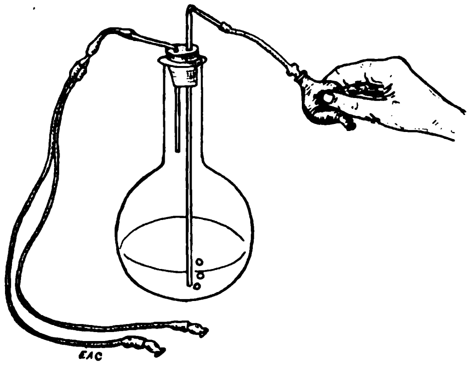


Fig. 108*B*.—The true mechanism of metallic tinkle.

cavities. *It never occurs in simple serous or purulent effusion, as no air is present.*

Succussion splash and metallic tinkle are the most pathognomonic signs of hydro-pneumothorax, but they only occur in about 30 or 40 per cent. of the cases (Cruice).

METALLIC TINKLE

This is a clear, vibrant, musical sound which may be heard in many cases of hydro-pneumothorax. It has been likened to the distant tinkling of a clear, high-pitched silver bell.

The genesis of metallic tinkle may be illustrated by the following experiment (Fig. 108). If we attach a stethoscopic tubing to the mouth piece of a wash bottle and listen while air is being forced through the other glass tube into the liquid, the phenomenon of metallic tinkle can be accurately reproduced. It will further be noted that the sound occurs at the moment the bubble is given off and not at the time at which it reaches the surface. If, on the other hand, we listen while water is allowed to fall from a pipette upon the surface of the water only a dull, indifferent sound will be heard.[1]

It is evident both from this and for anatomic reasons, that metallic tinkle is *due to air bubbles ascending through an effusion* from a fistulous tract in the lung below the level of the liquid, and not, as was formerly taught, to liquid dropping from a moist pleura upon the effusion beneath.

Metallic tinkle may also be produced by the bursting of bubbles in a bronchial tube which communicates with a pneumothorax or by a bubble in the surface of a moist perforated lung above the level of the fluid if the bubble is expelled with sufficient force. In either case the musical quality is due to the rhythmic vibration of the air, and the reverberation is due to the large air-filled, stiff-walled resonator—the pleural cavity (Fig. 106).

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[1] BARACH: *Arch. Diag.*, January, 1910.

CHAPTER XI

THE VOICE SOUNDS

The voice sounds consist of:

1. **Vocal resonance:** the voice sounds heard upon the chest.
2. **Bronchophony:** increased vocal resonance.
3. **Pectoriloquy:** bronchophony with articulated overtones—syllabic speech. Whispered pectoriloquy.
4. **Egophony:** a variety of bronchophony having a nasal or bleating quality.

These signs are elicited by auscultang over different areas of the chest wall, while the patient speaks or whispers. In the former case the patient should be told to slowly and loudly pronounce, "one—one—one," or "ninety-nine" in the lowest range of his natural speaking voice. The examiner should note any differences in clearness or intensity which may exist over symmetrical areas of the thorax.

VOCAL RESONANCE

The vibrations produced in the larynx during phonation are increased in intensity (larger wave amplitude) by (1) the thorax and to a minor degree by the trachea and bronchi, (2) by the air chambers above the vocal cords—the mouth and nares. These structures act as resonators, sympathetic vibrations being set up in them. But inasmuch as resonators act selectively, amplifying only certain wave lengths, the original sound as produced in the larynx undergoes certain changes not only in intensity but also in quality and pitch. *This explains why whispered pectoriloquy may be heard when vocal resonance is actually diminished.* Under normal conditions the vocal sounds heard over the chest are much less loud and clear than when we listen over the larynx. They seem distant and diffused, are lower in pitch, and the distinct articulations of the trachea are replaced by indistinct, rumbling sounds. The spoken word is lower in pitch when heard through the chest than when heard at the mouth, owing to the fact that the lung (resonator) which is large, reinforces the lower notes. When, however, the lung is consolidated, the sounds heard through the chest wall seem higher-pitched than at the mouth because the pulmonary tissue being infiltrated, only the bronchi remain to act as resonators, and also being small in size, only the higher notes are amplified (Mueller). It is thus evident that vocal resonance is due to somewhat the same causes, and is governed by the same laws as the breath sounds. Vocal resonance and vocal fremitus have the same origin. They generally vary in the same direction and in like proportions. Discrepancies between the two methods of examination may be due to the fact that the ear perceives vibrations better when they are rapid; the hand when they are slow. Vocal resonance may be (1) *increased*—more intense and clearer; (2) *decreased*—less intense and muffled. Here clearness is used in a double sense: (*a*) in regard to the fundamental note—the amount of sonorous laryngeal character; or (*b*) in regard to the articulated overtones—syllabic speech. These two qualities—clearness and loudness—do not necessarily run parallel, they may be diametrically opposed.

Normal Vocal Resonance.—Vocal resonance is normally most intense over those areas of the chest over which broncho-vesicular breathing is normally heard—the interscapular region, the upper sternum and the supraclavicular apices. But even here no distinct articulate speech can be appreciated; only an indistinct humming, buzzing, fluttering sound not unlike that of a voice heard at the distant end of a long hall. The sounds of both the spoken and the whispered voice are normally *more intense on the right side,* especially over the upper lobe, both anteriorly and posteriorly. This is due to the fact that the *right pulmonary apex lies in direct contact with the trachea,* and also because the right bronchus is almost a direct continuation of the trachea which points to the right (Fetterolf) (see Figs. 75, 95, 103).

In *women* vocal resonance is feeble because the fundamental note of the larynx does not stand in a favorable relationship with the thorax—the resonator. (Small tuning forks require small resonators.) In *men* the relationship is generally more favorable, although even here we meet with great degrees of difference. In singing an ascending scale from one's lowest base to one's highest treble, both vocal fremitus and resonance will become progressively less intense, and at a given point disappear entirely. *Children* often have a more intense fremitus than women because the aforesaid relationship is more favorable, although as a rule vocal fremitus and resonance are, in small children determined with difficulty (see p. 142). For the same reason these signs may vary with the degree of inspiration or expiration at which the patient speaks. If the sounds heard, remain intense even with marked stethoscopic pressure —which tends to blot out the sympathetic vibrations of the chest—we have even greater reason for believing that loss of elasticity and infiltration of the lungs has occurred (Sewall).

Vocal resonance may be *diminished* or absent in conditions which interfere abnormally with the conduction of vibrations from the larynx to the chest wall, such as obstruction of a bronchus, pleural effusions, edema of the chest wall or excessive subcutaneous fat. These conditions act through their effects of diffusion, reflection and resonance (see p. 60).

Vocal resonance is generally diminished over *pleural effusions* because vibrations are not readily transmitted from an air-bearing lung to the effusion. *If, however, the lower part of the lung which is in contact with the effusion, is solidified, either as the result of infiltration or compression, vocal resonance (as well as fremitus and breath sounds) will be intensely transmitted to the chest wall.*

Vocal resonance may be diminished in some rare cases of pulmonary fibrosis which would appear contradictory to what has been stated regarding normal and abnormal sound transmission, since solid lung, solid pleura and bronchus have relatively the same density and we should, therefore, expect what we usually find—increased voice and breath sounds. Montgomery suggests that diminished sounds may be due to (1) "heavy strands of fibrous tissue alternating with air-bearing lung or small cavities" thus offering a "break" in transmission; or (2) the fact that the bronchi are further removed from the chest wall by the thickened pleura and obliterated peripherally, or that their lumen is actually diminished.

Vocal resonance may, however, be diminished in some uncomplicated cases of pulmonary consolidation: (1) owing to bronchial obstruction due to (*a*) secretion (mucus, pus, blood, serum), (*b*) to pressure from without or (*c*) a malignant growth from within. In the first instance especially, the phenomenon may be temporarily dissipated by coughing or deep breathing. (2) Cases not explainable upon this basis may be due to the variable effects of resonance or wave interference already referred to (p. 55) (Montgomery).

BRONCHOPHONY

By this term we understand *vocal resonance increased in intensity and clearness.* Over normal pulmonary tissue the voice sounds are even more muffled and indistinct than when we listen near a primary bronchus. If the lung tissue is pathologically altered so that it becomes a better conductor of sound than is normally the case, we hear an increased vocal resonance—bronchophony.[1] In other words, the sounds are louder, higher-pitched, clearer and more amphoric than those we expect to hear in the locality in question, or than those we do hear in the corresponding area of the opposite (healthy) side.

Bronchophony, therefore, *occurs in:* (1) Pulmonary solidification—pneumonia, tuberculosis, atelectasis, compression, glandular enlargement especially if these conditions exist near to a large bronchus, etc. (2) Pulmonary cavitation—tuberculosis, abscess, gangrene, etc. In the latter instance the existence of bronchophony is in part due to the consolidation which accompanies and often surrounds the cavities. *Bronchophony is, therefore, generally encountered in association with increased vocal fremitus, bronchial breathing, percussion dulness and metallic or consonating râles.*

PECTORILOQUY

Pectoriloquy is *exaggerated bronchophony.* It is an increased vocal resonance in which syllabic speech (the articulated overtones) can be more or less distinctly recognized. In the ordinary speaking voice, as heard through the chest wall, the syllabics tend to be drowned out by the more intense and lower-pitched rumble of the vowel sounds, hence pectoriloquy can be better determined during the act of whispering.

The Whispered Voice Sounds.—Whispering is the result of articulate speech in which the glottis plays no part. Over the *normal chest* the whispered voice is scarcely audible, since whispering does not set up sympathetic vibrations of the thoracic wall. Over areas near to the primary bronchi one hears an indistinct swishing sound. **The transmission of syllabic speech**—*whispered pectoriloquy—is pathologic.*

Our judgment must be based not on the loudness but on the *distinctness of syllables.* The actual recognition of individual syllables is

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[1] Bronchophony as the derivation of the word implies, suggests the emanation of a sound from the bronchus. We receive the impression that the source of the sound is very near to the end of the stethoscope, or as if someone were talking directly into the latter. Some authorities speak of a "normal" and a "pathologic" bronchophony, using the former term as synonymous with vocal resonance.

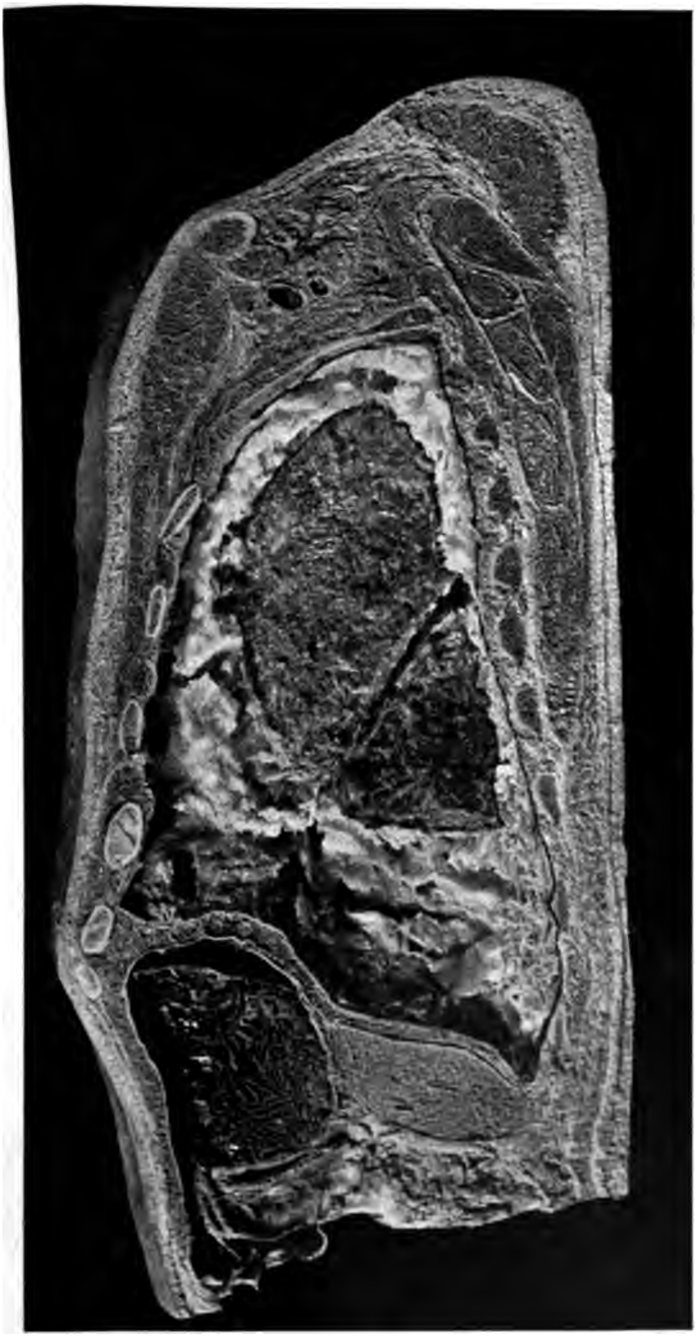

Fig. 109.—Hydrothorax. The lung, which is atelectatic, is surrounded by serum which ing frozen appears as ice crystals. The dome of the diaphragm is flattened and beneath it e seen a filled stomach and a portion of the left lobe of the liver.

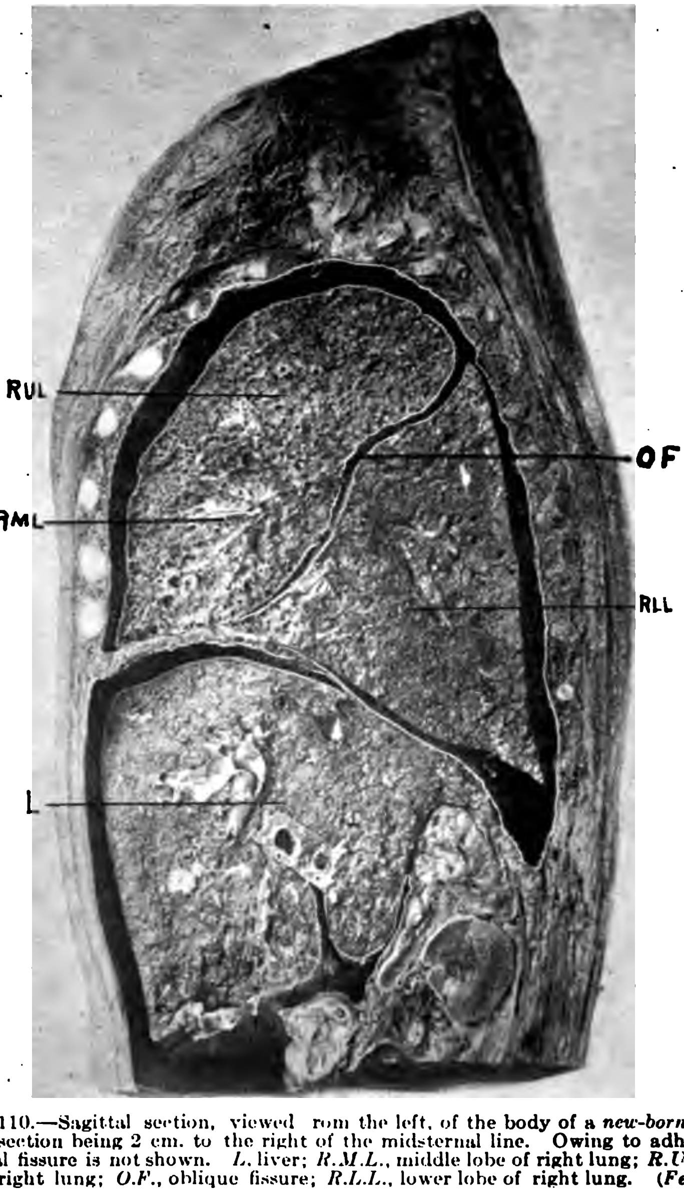

FIG. 110.—Sagittal section, viewed rom the left, of the body of a *new-born child*, the plane of section being 2 cm. to the right of the midsternal line. Owing to adhesions the horizontal fissure is not shown. *L.* liver; *R.M.L.*, middle lobe of right lung; *R.U.L.*, upper lobe of right lung; *O.F.*, oblique fissure; *R.L.L.*, lower lobe of right lung. (*Fetterolf and Gittings.*)

not a *sine qua non*. The sound is rarely sufficiently distinct to enable us to recognize every individual word.

The whispered voice is elicited by ausculting the chest while the patient whispers such words as "one," "two," "three," "sixty-six." Over the lung, soft, indistinct sounds are heard.[1] We hear "a feeble, low-pitched, blowing sound, these characters corresponding with those of the expiratory sound in forced breathing" (Flint). Sometimes no audible sound is produced. Near to areas of consolidation over which bronchial breathing is heard, the sounds become more distinct so that some syllables can be recognized, and over cavities or consolidations near a bronchus or cavities communicating with a bronchus, the acoustic impression is often nearly that of a person whispering directly into the stethoscope. In *women* and *children* the voice is higher-pitched and in the latter the large bronchi are nearer to the chest wall, thus the whispered voice is often more intense than in men. Whispered pectoriloquy is an important sign of pulmonary consolidation or cavitation. What has already been stated in regard to vocal resonance and bronchial breathing applies to it. It is much less taxing to the patient than speaking, and is often a useful method of determining pulmonary infiltration if a patient breathes so badly that bronchial breathing cannot be elicited. Whispered pectoriloquy is sometimes more distinct than spoken bronchophony, and may be elicited when the latter is absent, owing to the resonating properties of the tissues.

The whispered voice test further has the advantage over that of the spoken voice that does not set up sympathetic vibrations in the chest wall (resonator) and that an increase in intensity, therefore, speaks more strongly in favor of actual change in the pulmonary tissue.

The *whispered voice is normally heard* most clearly from the right apex to the second intercostal space. Posteriorly it is less intense and never extends below the scapular spine. On the left side its intensity is much less and its extent much more limited.

EGOPHONY

Egophony is a modified bronchophony characterized by a tremulous, high-pitched, bleating quality. It has been compared to the bleating of a goat, the speech of a person with a cleft palate, or to the voice of a ventriloquist. It may be imitated by attempting to throw the voice through the nares, while speaking with the nostrils occluded,[2] or by the interposition of a thin cork between the larynx and the stethoscope. It may be heard just below the upper level of pleural effusions while the patient is being examined for vocal resonance, and when present is a valuable sign of pleural exudation. It is perhaps due to the abnormal relationship between the bronchi (which become shorter when the lung is collapsed) and the compressed lung in pleural effusions that the necessary physical conditions are brought about which will reinforce the high-pitched nasal sounds.

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[1] Some people have never learned to whisper, some whisper during inspiration, which greatly modifies the sounds produced.

[2] Egophony was formerly attributed to (*a*) flattening of non-cartilaginous bronchi by pressure of the effusion, so that they acted like reeds (Laennec); (*b*) vibrations of the walls of small bronchi by actual collision—interruption of the air current (Wintrich); (*c*) the articulation of overtones—only the higher harmonics passing through the effusion (Stone); (*d*) to vibrations passing through a thin layer of fluid.

CHAPTER XII

THE PHYSICAL FINDINGS IN INFANTS AND YOUNG CHILDREN[1]

NORMAL CONDITIONS

Chest Inspection.—The antero-posterior diameter is relatively increased in infants (under two years). The sternum is prominent and the ribs more horizontal. *Breathing* is of the abdominal type for the first five or six years or even longer (see Fig. 17). The *chest wall* is thin and resilient and readily transmits sound vibrations. Auscultation should be practised before palpation or percussion, lest crying interfere with the former.

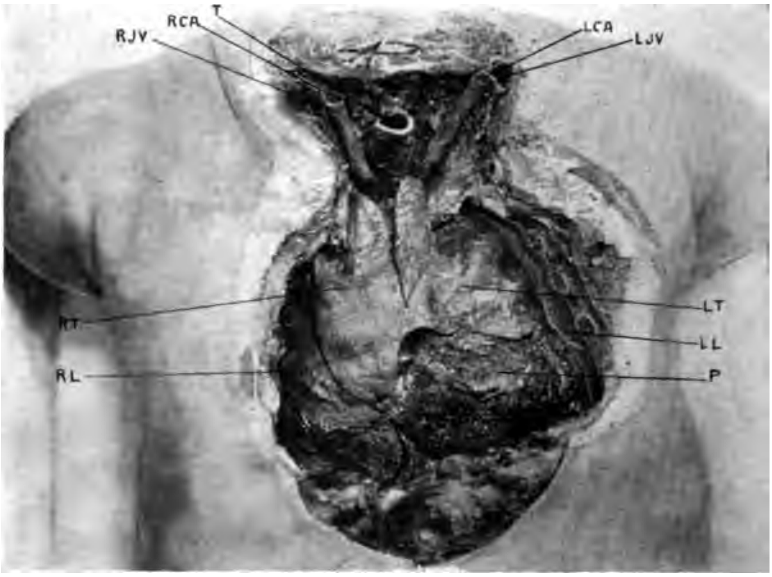

FIG. 111.—Body of a *new-born child* from which the median portion of the anterior chest wall has been removed. *R.L.*, right lung; *R.T.*, right lobe of thymus; *R.J.V.*, right internal jugular vein; *R.C.A.*, right common carotid artery; *T*, trachea; *L.C.A.*, left common carotid artery; *L.J.V.*, left internal jugular vein; *L.T.*, left lobe of thymus; *L.L.*, left lung; *P*, pericardium. (*Fetterolf and Gittings.*)

Palpation.—*Expansion* is slight and vocal resonance less marked than in adults (see vocal resonance). The *apex beat* is in the fourth interspace, just within or even to the left of the left mid-clavicular line.

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[1] For assistance in the preparation of the following paragraphs, we are indebted to DR. J. C. GITTINGS.

Percussion.—Percussion must be *extremely light*—finger percussion is often necessary. If a forcible stroke be employed the whole lung as well as the neighboring abdominal viscera will be thrown into vibration and topographic percussion will become impossible. The child must be placed in the sitting or standing position—or prone or supine—never on one side. Owing to the flexibility of the thoracic cage the lowermost lung when the infant lies upon one side becomes compressed and yields a less resonant note. The posterior portion of the thorax should be examined while the infant held in the nurse's arms, looks backward over her shoulder. The general percussion note is resonant, sometimes hyperresonant (higher pitched than in adults) from the clavicle to the fourth rib on right side anteriorly; to eighth rib posteriorly (on account of high dome of liver). Below these levels the note shades into liver dulness at the sixth and tenth ribs, respectively.

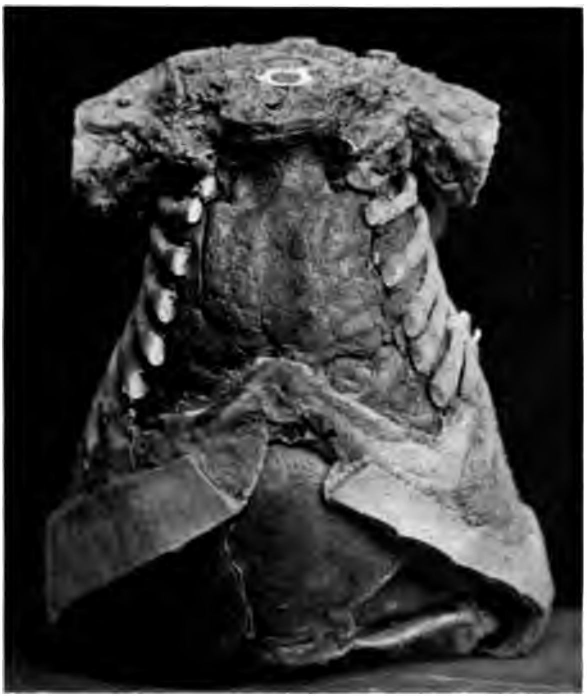

Fig. 112.—Thymus *in situ*. The anterior pulmonary margins are pushed aside. The lower border of the thymus gland overlies the greater portion of the heart. (*After Fetterolf and Gittings.*) (Compare Fig. 309.)

In the axilla and posteriorly on the left side *gastric tympany* may affect the normal note, as high as the sixth rib. This tympanitic quality may sometimes be eliminated or at least diminished by the method of lateral limitation—three fingers down, percuss the middle one.

On the *left side below the inner third of the clavicle* **the note is impaired or dull, shading into the area of cardiac dulness (Hamill), owing to poor expansion of left apex over the great vessels, possibly in part to left lobe** of thymus. *Crying* produces a rigid chest and may cause dulness over the lower lobes. Percussion during crying may yield a **cracked-pot sound** (p. 69). Percussion is generally less satisfactory than auscultation. The *sense of resistance* is often a valuable criterion in deciding between consolidation and liquid effusion.

The heart is large, lies higher in the thorax, as well as more horizontally. Cardiac dulness, therefore, normally extends relatively further to the left than in adults. It may normally extend ¼ inch beyond the mid-clavicular line. Dulness due to the right auricle, however, rarely extends beyond the sternal line. The great vessels are relatively large. This, together with the possibility of a persistent thymus gland renders a determination of the upper border difficult. This difficulty is further enhanced by the normal dull area at the inner third of the left clavicle already alluded to.

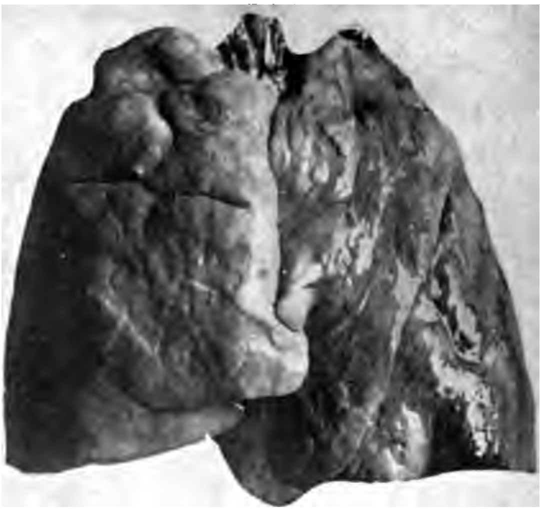

FIG. 113.—LUNGS OF A CHILD. In infancy and childhood the lungs often completely envelop the heart, there being in such cases no absolute (superficial) heart dulness.

During the first year the apex beat is usually found in the fourth intercostal space. This may be so until the thirteenth year, after which in normal children, it is always in the fifth interspace. As in adults, the existence of cardiac hypertrophy can usually be satisfactorily determined by inspection and palpation alone. If the left ventricle is chiefly involved, a heaving systolic thrust is felt outside of the mid-clavicular line. In case the right ventricle is chiefly affected, the thrust is diastolic in time and most marked in the epigastrium because the right ventricle retracts from the chest wall to rebound in diastole (Talley). Since compensatory and reparative changes in youth are good, marked degrees of cardiac hypertrophy are often seen in children with valvular lesions (Figs. 15, 122).

Cardiac Arrhythmia.—*Sinus arrhythmia*, or juvenile arrhythmia as it is often called, is as the latter name implies, very common in infants and children, especially in high strung youngsters and following infections (see p. 172).

Extrasystoles are also common, more so indeed in children with apparently normal hearts than in those with definite valvular lesions. *Tachycardia* is frequent after five years of age and *bradycardia* may occur during convalescence from fevers and in association with jaundice.

Auricular fibrillation is rarely met with and when encountered is usually only noted in fatal cases of diphtheria or rheumatic pancarditis.

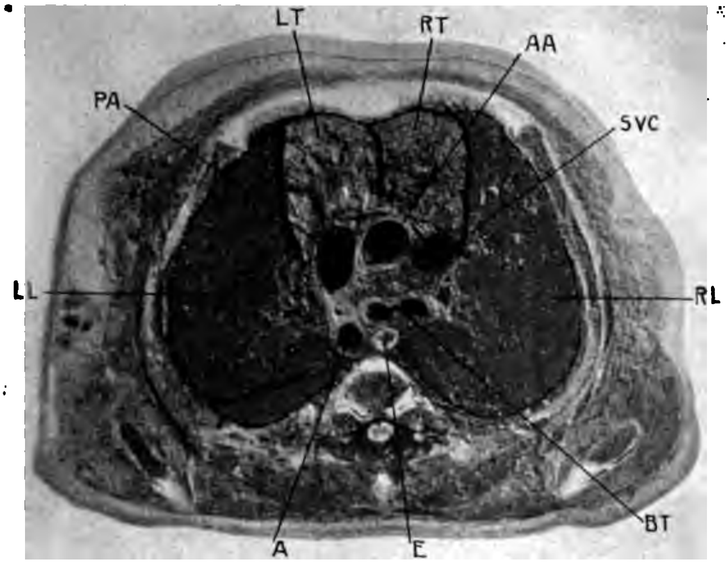

FIG. 114.—Horizontal section of a new-born *infant's thorax* at the upper level of the ensiform cartilage: showing its cylindrical shape. *L.L.*, left lung; *P.A.*, pulmonary aorta; *L.T.*, left lobe, and *R.T.*, right lobe of thymus body; *A.A.*, ascending part of aortic arch; *S.V.C.*, superior vena cava; *R.L.*, right lung; *B.T.*, bifurcation of trachea; *E.*, esophagus; *A.*, descending part of aortic arch. (*After Fetterolf and Gittings.*)

Auricular flutter is even more unusual, although Ritchie has reported two cases in children under ten years. *Heart block* also has been observed in association with diphtheria.

The prognostic significance of arrhythmia in children is essentially the same as in adults.[1]

Auscultation.—Respiration in infants and young children is irregular in rhythm and very irregular in depth. (Respiration being entirely a postnatal function, development of this function is relatively retarded. It does not become fully developed until the child has become active upon its feet. This early irregularity of breathing accounts in great part for the difficulty in interpreting the auscultatory phenomena.)

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[1] For more detailed information see TALLEY, J. E.: "The Physical Examination of the Heart in Children." *Arch. Pediatrics*, September, 1915, to which article I am indebted for some of the foregoing facts.

In early life, *the breath sounds* are relatively harsh, and loud. Expir tion is clearly heard, and in comparison with an adult relatively pr longed. Exaggerated or vesiculo-bronchial breathing is, therefore, normal finding (puerile breathing).

The areas overlying the bifurcation of the bronchi anteriorly extend ing 1 to 3 cm. to the right of the sternal margin, and 1 to 2 cm. to the lef (at level of the first interspace and the second rib) yield broncho-vesicula

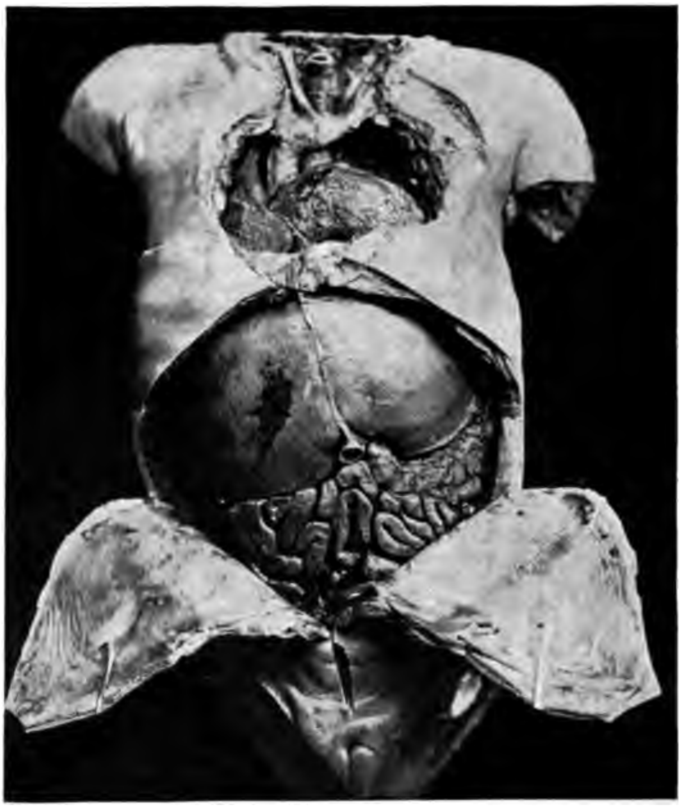


FIG. 115.—The *infant's chest* is circular in outline. The abdominal viscera are larg The proximity of the stomach and colon to the lower portion of the lungs imparts a tympa itic quality to the lower lobes, especially the left, and renders very light percussion essenti The heart lies almost horizontally, hence cardiac dulness extends relatively furter to th left than is the case in adults. It also lies about one interspace higher in infants.he

breathing. Posteriorly this is also found, especially on the right sid *Vocal resonance* in young children is hard to elicit as child cannot be con trolled. *Whispered pectoriloquy* can often be heard, normally, over th areas of broncho-vesicular breathing.

Owing to tardy development of the accessory muscles of respiratio and of the laryngeal and pharyngeal muscles, a slight hypersecretion o mucus in the pharynx, naso-pharynx or larynx, often produces coars

moist ***râles*** **which may be heard all over chest.** The ***heart sounds*** tend to be **embryocardial** in character, the first sound having a valvular quality, **and** the diastolic pause being brief. The pulmonic second sound is loud and more intense than the aortic (retracted lungs, more superficial position). Heart sounds and murmurs are loud.

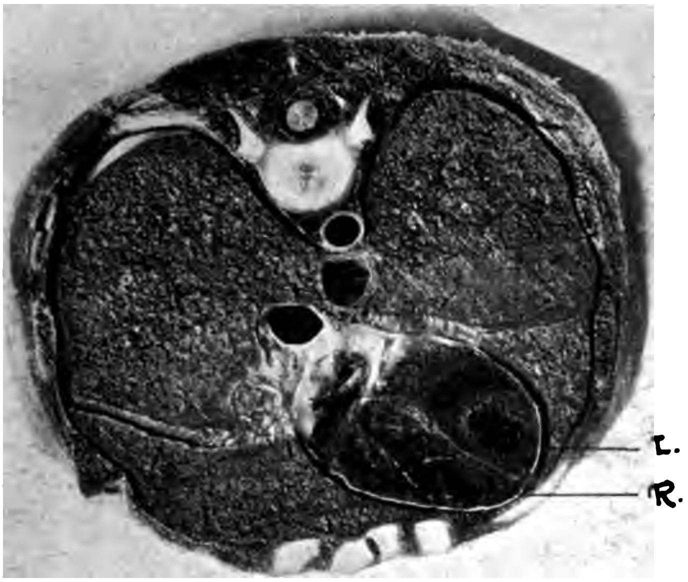


FIG. 116.—Horizontal section of an infant thorax showing its cylindrical contour as well as the relatively large size and horizontal position of the heart. *L.* = left ventricle. R. = right ventricle.

PATHOLOGIC CONDITIONS

Small *serous effusions* in the pleural cavities usually, but not invariably, produce a dull note on percussion. Large serous effusions may produce a dull or even a flat sound, as in adults, but quite often, a slightly tympanitic note results, due to relaxation of pulmonary tension and to abdominal tympany. The note over empyemas is more apt to be dull or flat, but, for the same reasons, this is not invariable. In serous or purulent effusions as a general rule, therefore, we do not find as flat a note as in adults, under the same conditions.

In ausculting over a **pleural effusion** (serum or pus) the **breath sounds often persist** and in some cases the diagnosis can be definitely established only by the exploring needle. In the case of *large effusions*, especially if purulent and causing marked compression of lungs, we may find bronchial or cavernous breathing, with whispered pectoriloquy, from the clavicle down as far as the third or even the fourth rib. An erroneous diagnosis of cavity is sometimes made. *Cavities in children are rare.*

The *second pulmonic sound* is actually and relatively louder than the second aortic—this difference persisting throughout childhood. *Organic*

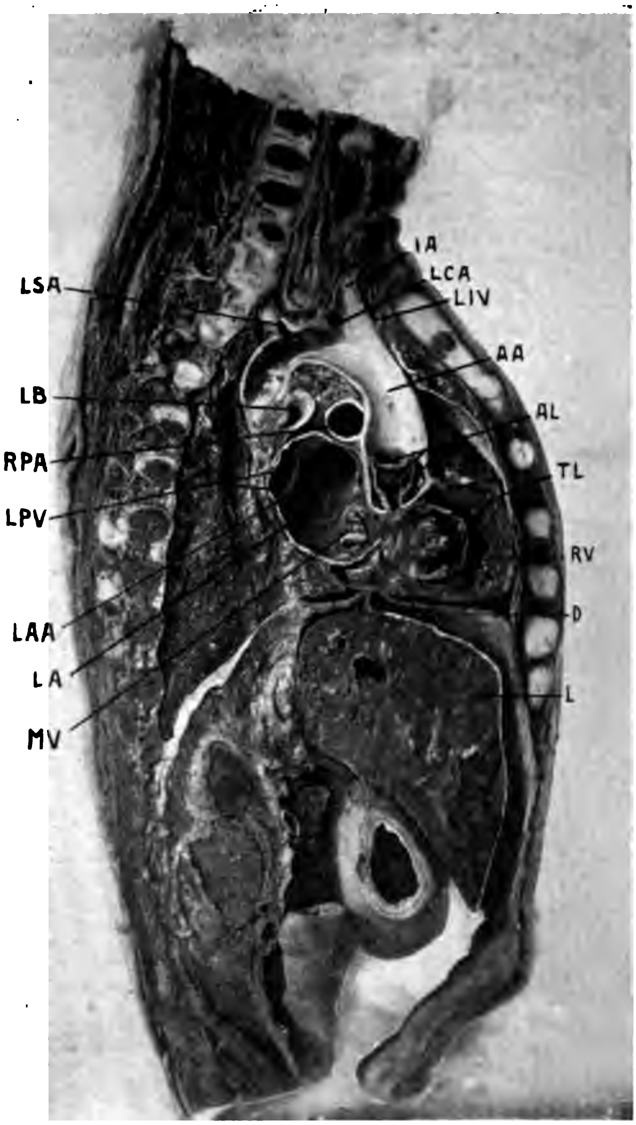
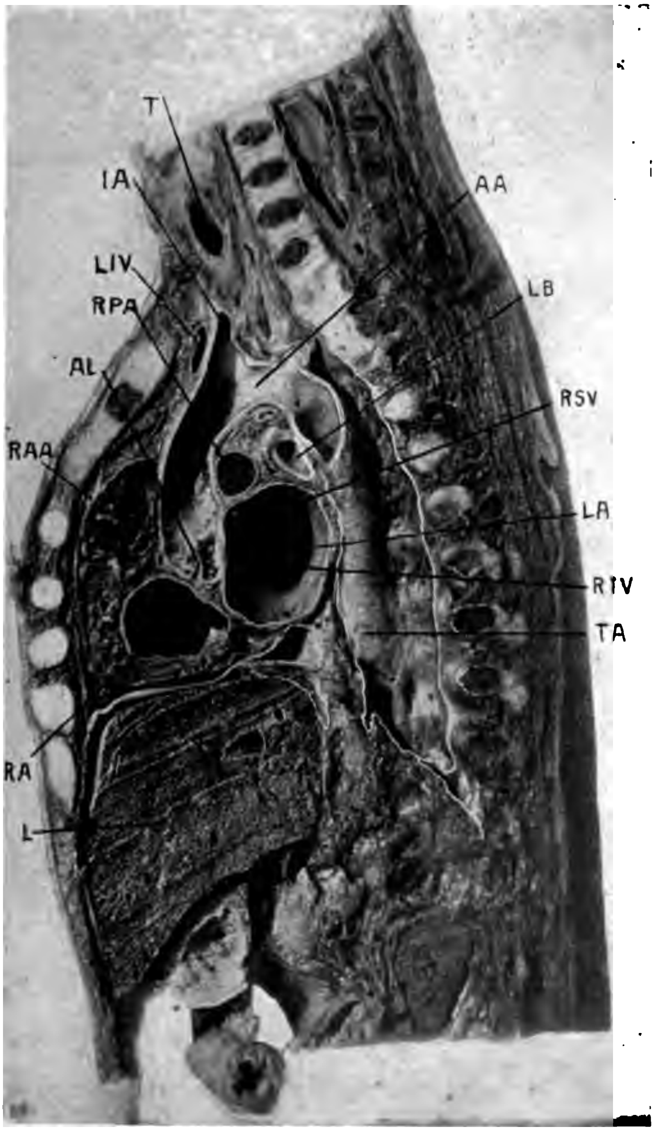


Fig. 117.—Sagittal section of the body of a *newly born child* viewed from the right, the plane of the section being 0.25 cm. to the left of the mid-sternal line. *M.V.*, mitral valve; *L.A.*, left auricle; *L.A.A.*, left auricular appendage; *L.V.P.*, orifices of the left pulmonary veins; *R.P.A.*, right pulmonary artery; *L.B.*, left bronchus; *L.S.A.*, left subclavian artery; *I.A.*, innominate artery; *L.C.A.*, left common carotid artery; *L.I.V.*, left innominate vein; *A.A.*, aortic arch; *A.L.*, aortic leaflets; *T.L.*, tricuspid leaflets; *R.V.*, right ventricle; *D*, diaphragm; *L*, liver.



118.—Sagittal section of the body of a *newly born infant, viewed from the left.* e of section being 0.75 cm. to the left of the mid-sternal line. *L*, liver; *R.A.*, right *R.A.A.*, left auricular appendage; *A.L.*, aortic leaflets; *R.P.A.*, right pulmonary *I.V.*, left innominate vein; *I.A.*, innominate artery; *T*, trachea; *A.A.*, aortic arch; bronchus; *R.S.V.*, right superior pulmonary vein; *L.A.*, left auricle; *R.I.V.*. left ulmonary vein; *T.A.*, thoracic aorta.

systolic murmurs, produced at the mitral orifice, usually can be heard clearly all over the area of the heart and posteriorly, on the left side. This is true to a lessened extent of aortic systolic murmurs in double aortic disease. *Aortic diastolic murmurs,* even more frequently than in adults, are heard best near the apex or at the fourth left costal cartilage.

Functional murmurs are very common in childhood and often simulate organic murmurs in quality, but are heard loudest over the base and in the reclining position. They become much less marked when the child sits up—an important distinction, since venous hums are intensified by this procedure.

Compression of the left lung by an enlarged heart or a pericardial effusion gives the signs of consolidation posteriorly—dulness and bronchial breathing—and must be differentiated from pneumonia.

In addition to the detailed anatomic relations the preceding figures illustrate the following facts:

1. The large amount of space in the antero-posterior direction occupied by the heart, enlargement of which (dilatation, pericardial effusion) would compress the upper part of the lower, and the lower part of the upper pulmonary lobes, causing bronchial breathing over the corresponding areas of the chest.
2. The superior vena cava enters the auricle at a plane posterior to that of the inferior cava, so that the blood stream from the latter is directed upward by the Eustachian valve and not to the left as is commonly stated.
3. The tricuspid orifice is practically vertical, not horizontal, and opens toward the left and slightly forward. The lower part of the right ventricle is so near the level of the right auricle that gravity can play but an inconspicuous part in the filling of the ventricle.
4. The mitral and tricuspid orifices lie in planes almost at right angles to each other.
5. The two posterior aortic leaflets are attached to the base of the mitral leaflet (see Flint's murmur).
6. The mitral, tricuspid and aortic valves are practically contiguous structures. This fact together with the actually small size of the heart, and the thinness of the chest wall, explains why sounds produced at these areas are often differentiated with difficulty. A systolic aortic murmur can only be identified with certainty if heard in the neck.[1]

PRACTICAL CONSIDERATIONS

In examining the lungs, the chest must be exposed and the patient must not be in a recumbent position if the greatest possible accuracy is a desideratum. The importance of inspection, especially in chronic pulmonary disease, cannot be over-emphasized. Symmetric areas of the chest must be examined and compared step by step. In determining fremitus it is well to bear in mind that the ulnar nerve distribution is more sensitive than the radial and hence the hand should be slightly rotated toward the outer side.

In practising percussion the beginner usually employs far too much force. This is always disagreeable and often painful to the patient; it is also hard on the examiner's fingers. As a general rule, the more experienced the examiner the lighter the percussion stroke he employs. Force and accurate limitation are not compatible. The blow must be delivered from the wrist (not the arm), it must fall vertically and the striking finger must be quickly withdrawn. Good percussion is, and should be, a "gentle art."

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[1] Fetterolf and Gittings: *Am. Jour. Dis. Children,* I, 1911, 6.

Before ausculting, see to it that the patient breathes properly. Respirations should be a trifle deeper than is normally the case, and the mouth slightly open. "Throaty" sounds must be eliminated and expiration must be purely passive. The areas over which the breath sounds may normally be somewhat harsh, must be borne in mind, and examination of the supraclavicular and suprascapular portions of the lung as well as the apex of the axilla must not be omitted.

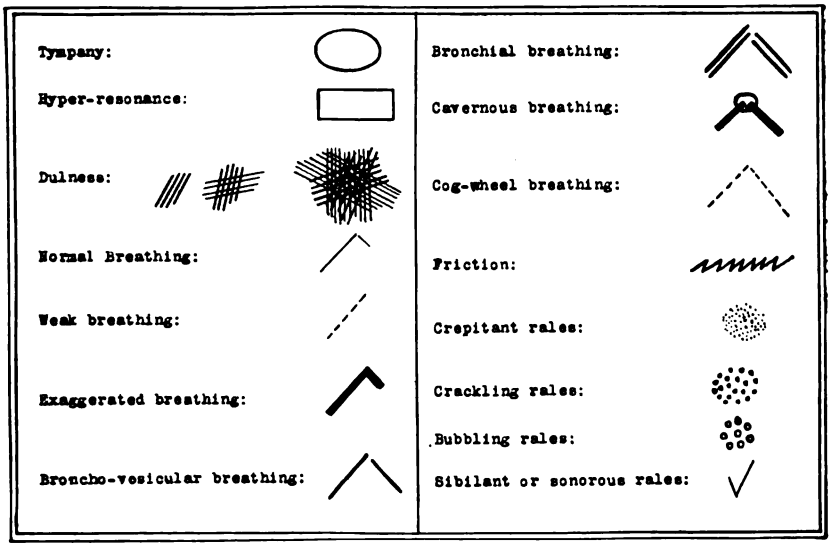
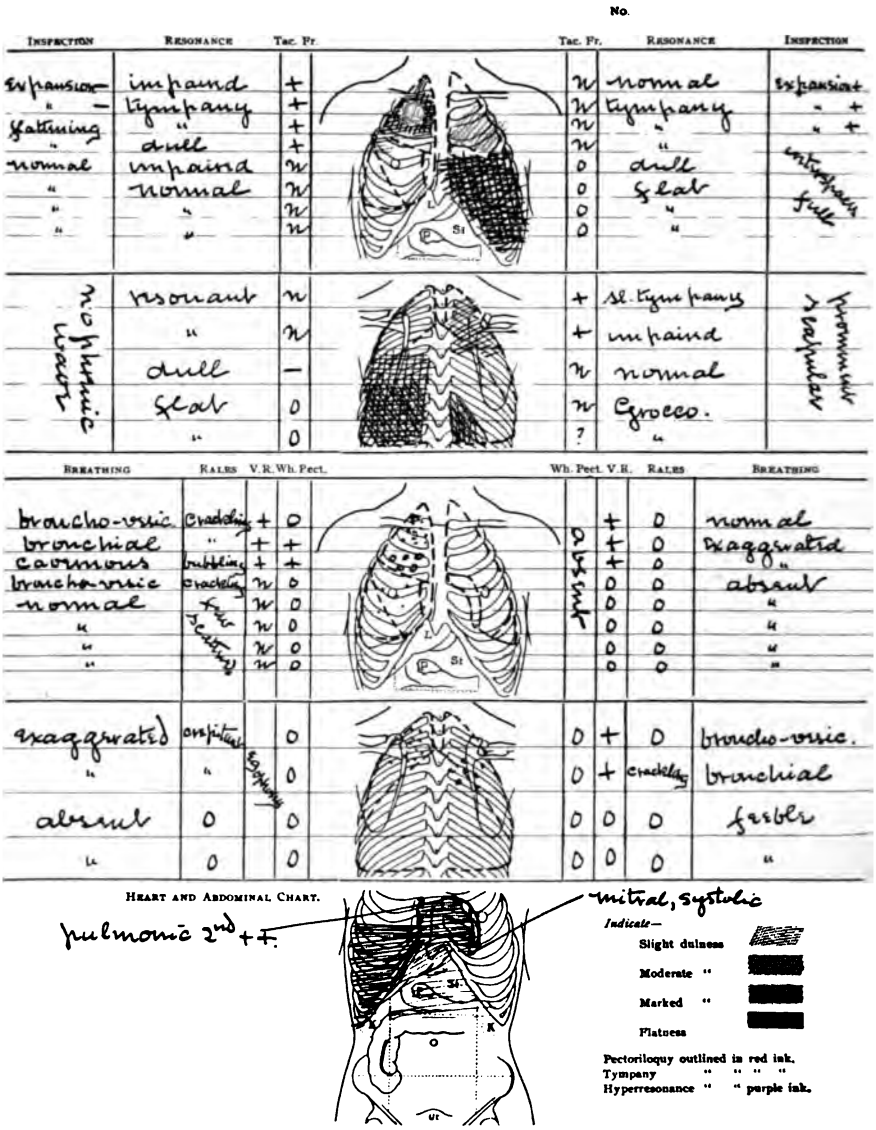


Fig. 119.—For the sake of conserving time in the recording of physical signs various systems of graphic registration have been suggested. That which follows is modified after Barach's plan. Unfortunately no uniform system has thus far been adopted by clinicians.

If rôles occur during inspiration they are charted before, if during expiration, after the symbol which stands for the type of breathing.

A diagnosis of incipient tuberculosis based upon a single examination is always a questionable procedure. Repeated examinations and further observation are usually advisable. In really early pulmonary tuberculosis, abnormality in the physical signs is very slight and may be absent. A careful consideration of the patient's history and an observation of his temperature and pulse are often more reliable than slight abnormality of his physical signs.

The physical signs of pleural effusion and pulmonary consolidation are variable, and the classical text-book method differentiation often fallacious. Consolidation may yield absent breath sounds; an effusion, bronchial breathing, bronchophony and fremitus. The reasons for this



No.

Inspection	Resonance	Tac. Fr.	Tac. Fr.	Resonance	Inspection
Expansion –	impaired	+	n	normal	expansion +
" –	tympany	+	n	tympany	" +
flattening	"	+	n	"	" +
"	dull	+	n	"	intercostal spaces full
normal	impaired	n	0	dull	
"	normal	n	0	flat	
"	"	n	0	"	
"	"	n	0	"	

Inspection	Resonance	Tac. Fr.	Tac. Fr.	Resonance	Inspection
no phthisic habit	resonant	n	+	sl. tympany	prominent scapulae
	"	n	+	impaired	
	dull	–	n	normal	
	flat	0	n	Grocco.	
	"	0	?	"	

Breathing	Rales	V.R.	Wh. Pect.	Wh. Pect.	V.R.	Rales	Breathing
broncho-vesic.	crackling	+	0		+	0	normal
bronchial	"	+	+	absent	+	0	exaggerated
cavernous	bubbling	+	+		+	0	"
broncho-vesic	crackling	n	0		0	0	absent
normal	few scattering	n	0		0	0	"
"		n	0		0	0	"
"		n	0		0	0	"
"		n	0		0	0	"

Breathing	Rales	V.R.	Wh. Pect.	Wh. Pect.	V.R.	Rales	Breathing
exaggerated	crepitant	egophony	0	0	+	0	broncho-vesic.
"	"		0	0	+	crackling	bronchial
absent	0		0	0	0	0	feeble
"	0		0	0	0	0	"

HEART AND ABDOMINAL CHART.

pulmonic 2nd ++.

mitral, systolic

Indicate—
Slight dulness ▨
Moderate " ▩
Marked " ■
Flatness ■

Pectoriloquy outlined in red ink.
Tympany " " " "
Hyperresonance " " purple ink.

FIG. 120.—The accompanying chart, which is the type used at the Phipps Institute, depicts the physical signs encountered in a case of right-sided pulmonary tuberculosis with cavitation in the upper lobe, and of left-sided pleural effusion. In addition to furnishing excellent graphic records the details of which can be grasped at a glance, the method is invaluable for students since they are required to describe exactly the signs occurring in every region of the chest. This encourages care for detail and eliminates "negative" results. Red shading indicates tympany.

variation of physical signs have already been discussed (p. 60, Fig. 107). The exploring needle should be unhesitatingly employed in doubtful cases. Properly performed, this procedure is unattended by risk, or any considerable degree of pain.

The explanation of abnormal physical signs as "thickened pleura" should be made only as a last resort. In this condition physical signs are slight or entirely absent, unless the lung is also involved.

With obscure pulmonary symptoms, especially in men past thirty-five years, the possibility of aortic aneurism must ever be borne in mind, especially if associated with cough, chest pain, or dyspnea for which symptoms no evident cause can be demonstrated.

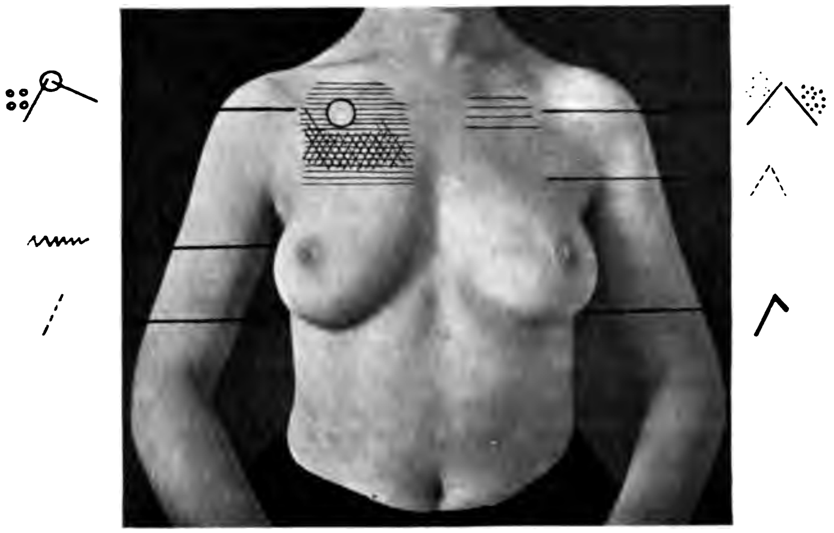


Fig. 121.—The graphic signs here depicted would be construed as follows: *Right upper lobe:* high-pitched tympany, surrounded by dulness, cavernous breathing, inspiratory bubbling râles. *Right axilla:* friction and below this area weak breath sounds.

Left upper lobe: slight dulness, broncho-vesicular breathing, subcrepitant inspiratory and crackling, expiratory râles. *Left axilla:* cog-wheel breathing, below this area, exaggerated breath sounds.

THE X-RAY

The X-ray, especially as a fluoroscope, has become a useful adjunct in intrathoracic diagnosis. It enables us to "control" our percussion and often definitely establishes the presence or absence of deep-seated mediastinal lesions—aneurism, lymphadenitis, central pneumonia, tumors, foreign bodies, mediastinal displacement, pulmonary abscess, etc. —in cases in which ordinary physical diagnosis only permits us to "suspect" the lesion. The same statement applies to some cases of localized pneumothorax and of pulmonary fibrosis involving the diaphragmatic pleura, as well as to small pleural effusions.

On the other hand, the ordinary methods of diagnosis are far superior to the X-ray in cases of incipient or early pulmonary tuberculosis and in congestion of the lungs. In these instances the mistakes are usually on the side of the radiographer. The X-ray is rarely of much use as an early diagnostic method in cardiac or pulmonary disease.

PART II

THE EXAMINATION OF THE CIRCULATORY SYSTEM

By George W. Norris, A. B., M. D

CHAPTER XIII

THE CIRCULATORY SYSTEM

INSPECTION

The most important signs to be looked for are: dyspnea, orthopnea, pallor, flushing or cyanosis; distention or pulsation of the superficial arteries or veins, especially in the neck; edema, especially of the dependent portions of the body and of the face; clubbing of the fingers, inequality of the pupils; normal or abnormal pulsations, especially of cardiac apex and the epigastrium; distention of the abdomen, fulness, in the hepatic region, and in the flanks.

Orthopnea, the state in which the patient has to sit up in order to get his breath—indicates that the heart is drawing on its last reserves. *Circulatory pallor* is seen in connection with low blood-pressure and peripheral anemia. *Flushing* of the face may be seen in fevers, excitement also in aortic insufficiency and at times in arterial hypertension. *Cyanosis* indicates insufficient oxygenation of the blood, and is seen chiefly in mitral, pulmonary and tricuspid lesions associated with pulmonary congestion. It is most marked in the face, hands, and feet. *Distention of the superficial veins* indicates high venous pressure, generally a weak right heart; or local obstruction to the venous circulation and an effort to establish collateral circulation. *Circulatory edema* occurs characteristically at the end of the day, and in the dependent portions of the body; *renal edema* occurs in the morning and upon the face. *Clubbing of the fingers* indicates long-standing stasis of the pulmonary circulation—chronic cardiac or pulmonary disease. *Anisocoria*—pupillary inequality—may result from pressure of a thoracic aneurism upon the cervical sympathetic. *Pulsation of the cervical vessels* may be arterial or venous. In the former instance it may be due to marked alternations of blood-pressure and an hypertrophied heart, or to a local aneurism. Venous pulsations may be seen in the jugular veins of recumbent subjects. Normally three small, faint waves can be made out. These are stasis waves; they are not due to regurgitation from the right heart. A single large systolic pulsation may result from (*a*) a transmitted impulse from a contiguous artery, (*b*) tricuspid insufficiency, (*c*) aneurismal varix. A *capillary pulse* (Quincke's pulse) consists in an alternate flushing and blanching of a finger tip when slight pressure is made upon the lower portion of the finger nail. It may occur in cases of aortic insufficiency. It is not an early sign and is elicited with difficulty.

The patient who suffers from *mitral* and *tricuspid disease* is apt to complain of orthopnea, cough, cyanosis, and hemoptysis (pulmonary stasis), digestive disturbances (portal congestion) and edema. ***Aortic lesions*** are characteristically associated with dyspnea on exertion, vertigo, flushing, palpitation, precordial oppression.

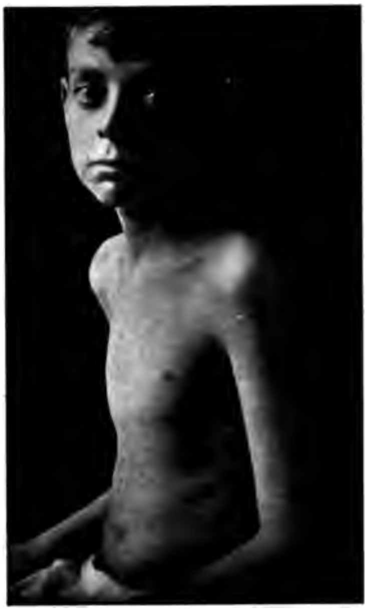


FIG. 122.—Stunted growth and bulging of the precordium resulting from **mitral obstruction** and insufficiency in a boy of fifteen years.

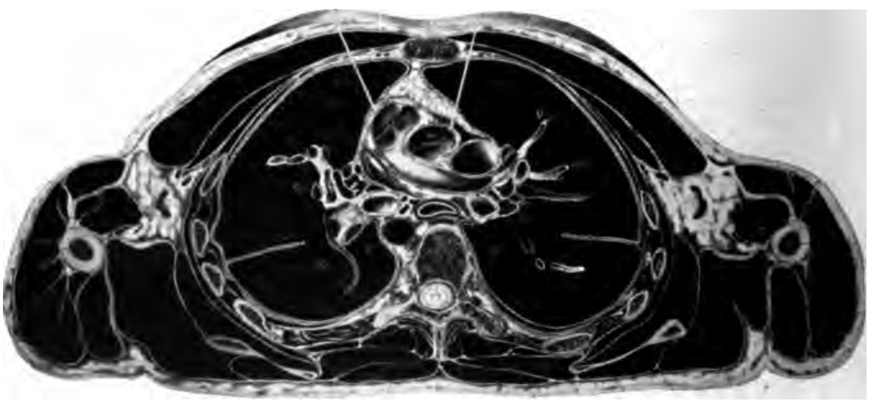


FIG. 123.—Showing partial overlapping of the aortic and pulmonic valvular orifices. The pulmonic is the most superficial of all the cardiac valves.

THE HEART—ANATOMICAL CONSIDERATIONS

The heart lies obliquely placed behind the lower two-thirds of the ternum. It rests upon the upper surface of the diaphragm, two-thirds f its bulk being placed to the left of the mid-sternal line. The *base* exends from the lower border of the second left costal cartilage to the upper

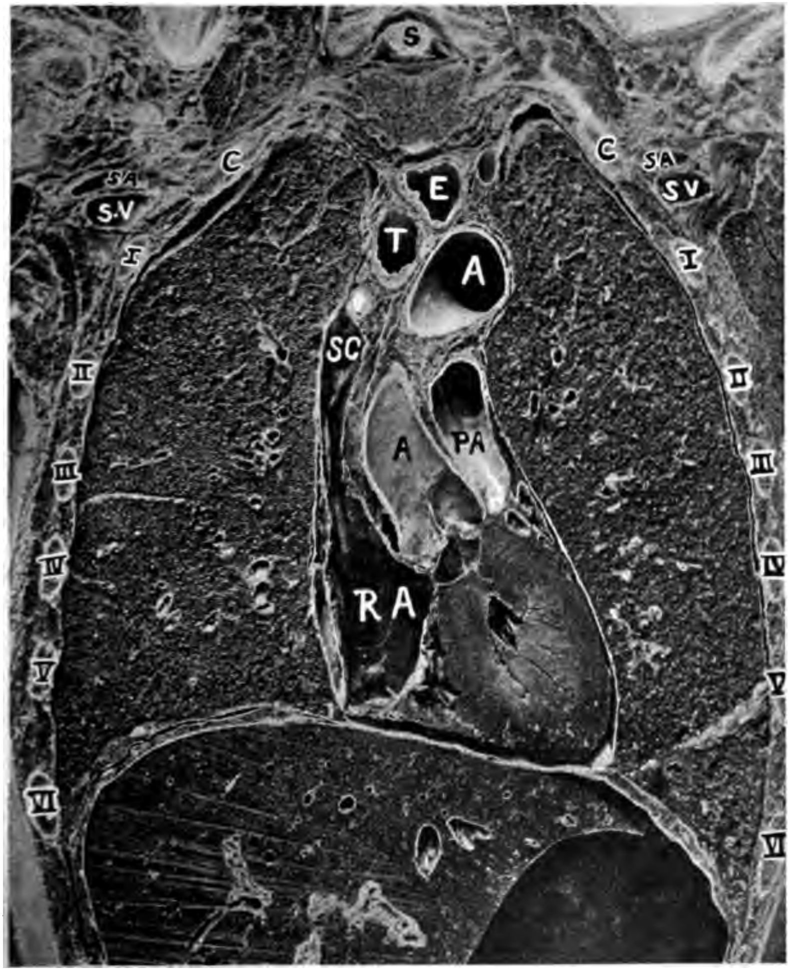


Fig. 124.—Normal position of the heart. *A.* = aorta; *P.A.* = pulmonary artery; .*A.* = right auricle; *S.C.* = superior vena cava; *T.* = trachea; *E.* = esophagus; *S.A.*, sub-avian artery; *S.V.* = subclavian vein; *S.* = spinal cord; *C.* = clavicle.

order of the third right costal cartilage. The *apex* lies in the fifth left itercostal space, about 8 cm. to the left of the mid-sternal line. The *orta* arises at the level of the third costal cartilage behind the sternum. ; reaches halfway up the manubrium, and is nearest the surface at the econd right costal cartilage.

The Valves.—The *aortic valve* lies behind the third left costo-sternal junction. It is one-fourth overlapped by the pulmonary valve. The *pulmonary valve* lies a little higher and more to the left. The *mitral valve* lies behind the third intercostal space about 1 inch to the left of the sternum. The *tricuspid valve* lies below and slightly to the left of a line drawn from the inner end of the third left, to the sixth right costal cartilages. The aortic, mitral and tricuspid valves are anatomically practically contiguous structures. The mitral is the furthest from, the pulmonic the nearest to, the anterior chest wall (Figs. 123, 128).

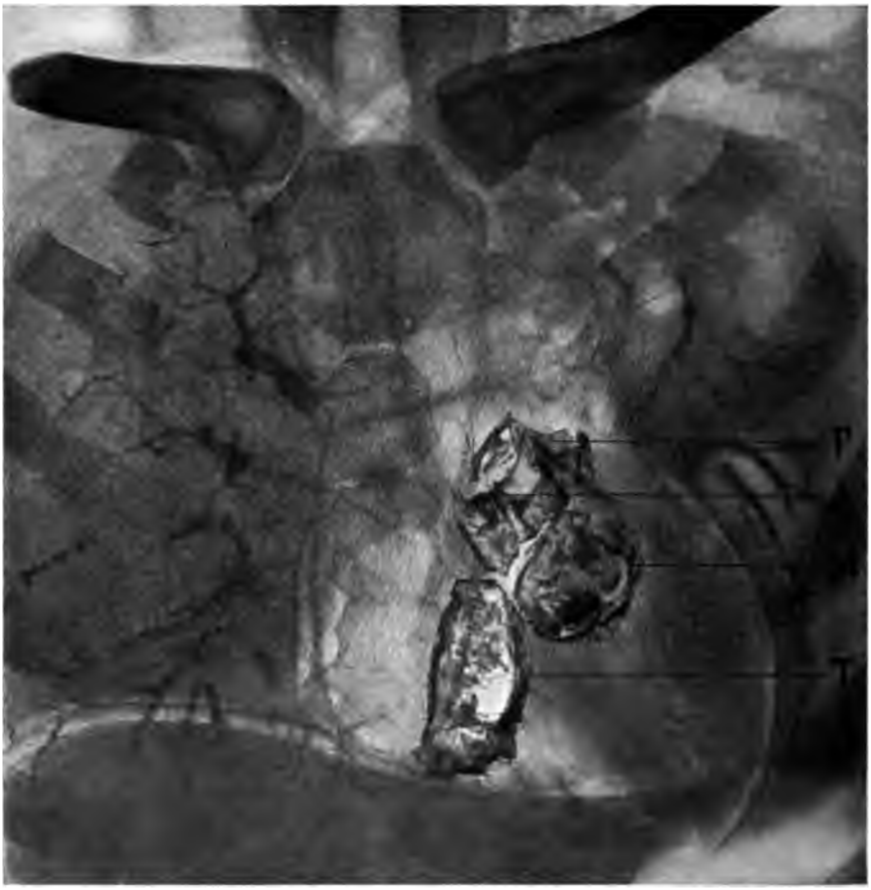


FIG. 125.—Radiogram showing the *relative position of the cardiac valves* in relation to each other and to the anterior bony thorax. The pulmonary, in part overlies the aortic, valve. It is the most superficially placed of all the heart valves. The mitral valve is the farthest from the anterior chest wall. The large size of the tricuspid orifice is well shown. This valve is structurally a much less perfect mechanism than is the mitral.

The valves were covered with lead paint before making the radiogram. (*After Norris and Fetterolf.*[1])

Posteriorly.—The base of the heart lies at the level of the fifth dorsal

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[1] NORRIS and FETTEROLF: "The Topography of the Cardiac Valves as Revealed by the X-rays." *Am. Jour. Med. Sc.*, cxlv, 1913, 225.

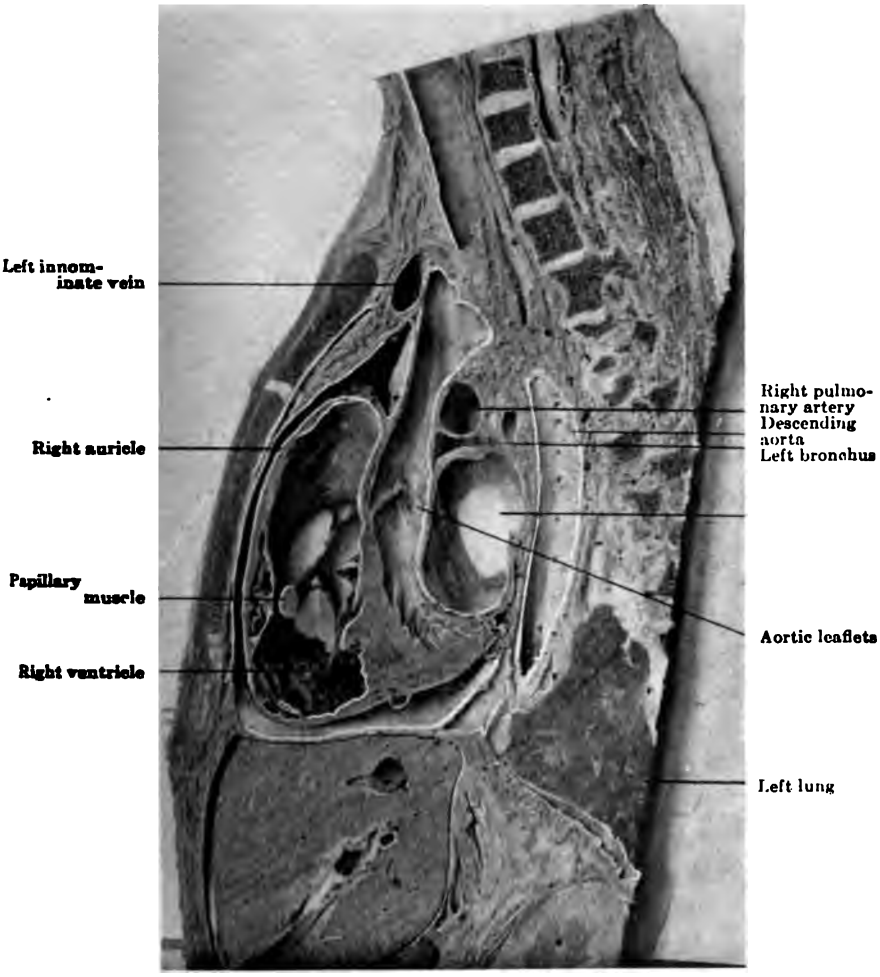
Left innominate vein
Right auricle
Papillary muscle
Right ventricle
Right pulmonary artery
Descending aorta
Left bronchus
Aortic leaflets
Left lung

Fig. 126.

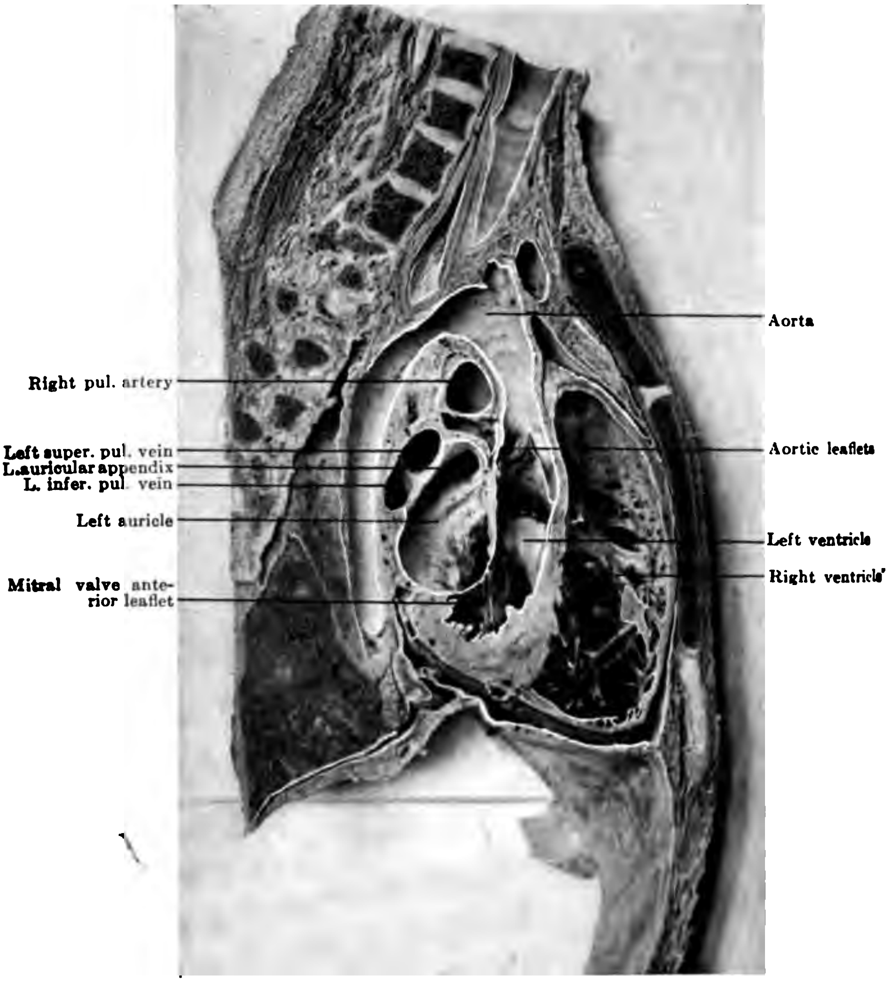

FIG. 127.

FIGS. 126 AND 127.—These sections show the deeply placed position of the left auricle which may be topographically described as the "posterior auricle." They also depict the relatively large amount of mediastinal space which is occupied in an antero-posterior direction by the heart. The rear view of the heart is shown in Figs. 91 and 92. the anterior aspect in Figs. 86 and 163.

vertebra. The apex of the heart lies at the level of the eighth dorsal vertebra. The aorta reaches the spine at the level of the fourth dorsal vertebra. The pulmonary artery bifurcates at the level of the fourth dorsal vertebra. Practically the whole anterior surface of the heart is right heart (see Fig. 163). The left auricle lies posteriorly. Of the left ventricle only the tip is seen from in front.

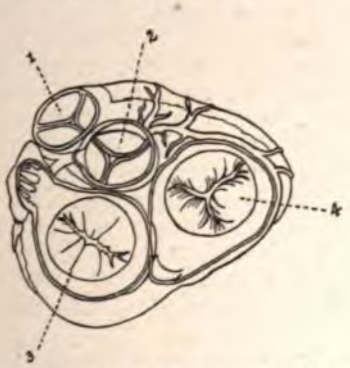

FIG. 128.—Diagram illustrating the relative position of the heart valves as seen from above: 1, pulmonary; 2, aortic; 3, mitral; 4, tricuspid. The pulmonary is the most superficial, and the mitral the most deeply placed of the cardiac valves (comp. Fig. 123).

PALPATION OF THE PULSE

The pulse is generally examined by palpation of the radial artery. When the patient's hands are not accessible, as during sleep, the temporal artery is often selected. Under certain circumstances it may be necessary to feel the pulse elsewhere as, for instance, in the tibial arteries or in the dorsalis pedis. The pulse should be palpated with the tips of the fingers, not with the palmar surface. The thumb must not be used because a subjective pulse can often be felt. When examining the pulse two conditions are to be determined: (1) *the character of the arterial wall* (arterio-sclerosis, etc.); and (2) *the character of the pulse.*

1. **Arterial thickening** is determined (*a*) by rolling the emptied artery, in which the blood flow has been temporarily occluded by pressure above and below the point of palpation, under the ball of the finger; (*b*) by allowing the end of the finger nail to slip across the arterial wall. Normal arteries are barely palpable.

An important source of error, however, lies in the state of vascular tonus. When blood-pressure is high the artery may readily be rolled under the finger, and the tactile sensation received may be similar to that produced by actual arterial sclerosis. Increased tonus may sometimes be temporarily abated by local massage of the artery. On the other hand, a definitely thickened artery may escape detection when tonus is diminished.

If arterio-sclerosis is suspected, examination must not be limited to the radial arteries but should include the brachials, femorals, temporal and retinal arteries as well.

2. **The character of the pulse** is judged by noting the *rate, rhythm, volume, tension and equality.* Both radial arteries should be habitually palpated synchronously in order to determine bilateral equality.

The Pulse Rate.—The normal rate in adults is for men 72, for women 80. With advancing years the rate often becomes slower. Certain individuals in perfect health may have by nature a rapid (90) or a slow (60–70) pulse. The latter rate is not infrequent in advanced years. In the new-born the rate averages about 140, and at fourteen years about 90. The pulse rate is increased by excitement, anger, fear, exercise, digestion, deep inspiration, low blood-pressure, fever, etc. The pulse increases about ten beats for every degree Fahrenheit above the normal body temperature. It is more rapid in short, high-strung individuals than in people with a large frame and a phlegmatic disposition. The peripheral pulse rate does not always indicate the cardiac rate. Notable discrepancies may occur in auricular fibrillation, extrasystolic arrhythmia and pulsus alternans. The complete bodily circuit of blood flow requires normally about 23 seconds and is accomplished by about 27 systoles. The pulse wave travels 9 to 10 m. per second, it therefore reaches the dorsalis pedis artery ⅙ second later than the aorta—about the middle of ventricular systole. The time required to reach the radial artery is approximately 0.17, and the interval between the carotid and the radial pulses, is 0.08 second.

Tachycardia.—This term is generally applied to a pulse rate above 130 per minute. It may occur in exophthalmic goitre, in cardiac disease, in vasomotor collapse, etc., but is most characteristically seen in "paroxysmal tachycardia," a condition due either to extrasystoles or to a fibrillating auricle.

Bradycardia.—This term is generally applied to a pulse rate of, or of less than, 60 per minute. It may occur in convalescence, after vomiting, in basilar meningitis or increased intracranial pressure (vagus irritability or stimulation) and also in jaundice (toxic effect). It occurs characteristically in heart block, in which condition it may fall to twelve beats per minute.

The Pulse Rhythm.—The pulse may be irregular either in regard to (1) time or (2) force. Not infrequently both factors are combined, as in auricular fibrillation (see Cardiac Arrhythmia).

The Pulse Volume.—The volume of the pulse is gauged by estimating the degree of pulsatile oscillation—the amount of systolic filling and the completeness of diastolic collapse. It depends upon, and is in part a measure of the systolic output. The *pulse pressure*—the difference between the systolic and the diastolic pressures—may be taken as a rough index of pulse volume. As a general rule the more rapid the rate, the smaller the volume of the pulse.

The Pulse Tension.—This is estimated by gauging the amount of pressure which must be exerted upon the artery before the flow of blood is stopped. In other words it depends upon the blood-pressure which is due chiefly to vascular tonus, cardiac force and valvular sufficiency. The pulse is sometimes described as hard or soft, depending upon whether arterial tension is high or low. These terms are obsolete, the degree of tension should be measured with a sphygmomanometer and the height of the systolic and the diastolic pressures recorded in millimeters of mercury (see Blood-pressure Instruments, p. 163). The celerity of the pulse wave depends upon the suddenness of its collapse. A "*quick*" *pulse is not a rapid pulse,* but one which disappears quickly. It occurs characteristically in aortic insufficiency and is known as the water-hammer or Corrigan pulse, but it also occurs in a minor degree in severe anemia and in vasomotor paresis.

The opposite condition—*pulsus tardus*—appears and disappears slowly. The systolic ascent of the sphygmogram is gradual, the plateau long and the descent prolonged. This type of pulse is observed especially in aortic, and in a minor degree mitral, obstruction. It is a valuable diagnostic sign of the former.

The Equality of the Pulse.—By this we mean equality in (1) volume, (2) time and (3) force, in symmetrical arteries of the body. *Inequality of the radial arteries* may result from: (1) surgical injuries of the upper extremities, (2) axillary growths, (3) *aneurism of the aorta*, innominate, subclavian, brachial or radial arteries, (4) emboli or thrombi or syphilitic disease in these arteries, (5) massive pleural effusions, (6) local inflammatory conditions, (7) extreme auricular dilatation. Slight degrees of inequality occur very commonly. This sign is of little importance unless it is well marked.[1]

NORMAL AND ABNORMAL TYPES OF THE ARTERIAL PULSE

The normal arterial pulse which is felt as a single "beat" consists when analyzed of the following waves:

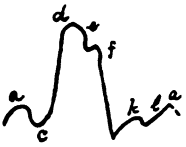

FIG. 129.—The cardiogram (tracing from the precordium): *a*, auricular wave; *c.d.-e.f.*, ventricular systole); *d*, marks the opening and *f*, the closure of the semilunar valves, (beginning of diastole); *k*, sudden ventricular filling.

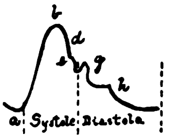

FIG. 130.—The normal radial curves: *a–b*, the tidal wave; *b–e*, the aortic notch; *g*, the dicrotic wave; *h*, the post-dicrotic wave.

The arterial pulse, by which term we designate the expansion and **contraction** of an artery which occurs during the systole and diastole of **the heart, has** long been used as a diagnostic criterion. When the sphygmograph **was** first introduced in medicine it was hoped that a detailed **study** of the length, height and time relations of the various phases of **the pulse** tracing would lead us far toward perfection in diagnosis. As a **result** of such studies many different types of the arterial pulse were **classified**, named and studied, though with but little practical result. **Such studies** do, of course, enable us to visualize our palpatory sensations, **but the** value of the sphygmogram to-day lies almost wholly in the time **relationship** between the arterial and the venous pulse waves. There **are, however,** certain types of the arterial pulse which are more or less **characteristic** of certain conditions which find a place in clinical nomenclature and are recognizable by the sense of touch. Among these the following may be mentioned:

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[1] In a study of 500 cases (well and ill) the two radial pulses were alike in only 56 per cent. There was marked difference in 2.5 per cent. The left pulse was stronger in 14 per cent. of 455 right-handed people, and in 43 per cent. of the 21 ambidextrous. In advanced life inequality is generally due to arteriosclerosis (KOENNICKE: *Therap. d. Gegenw.*, September, 1911).

The Dicrotic Pulse.—A dicrotic pulse is one of low tension in which the normal secondary wave becomes exaggerated, so that it may be both felt and instrumentally demonstrated to consist of a more or less separate wave. It appears therefore as a repetition or echo of the primary wave. If the pulse rate is rapid, the line of descent of the dicrotic wave is interrupted by the next primary wave, which condition is known as *anacrotism.* In the majority of the febrile cases at least, it is brought about by relaxation of the arteries and constriction of the arterioles, which condition causes an actual backward flow of blood in the primary pulse wave, a phenomenon which is repeated in the case of the dicrotic wave. Other causes such as a brief systolic output from the heart, or dilatation of certain vascular areas, especially the splanchnics, have been suggested (Hewlett).

The Pulsus Bisferiens.—This term should be applied only to those cases in which the apices of the two waves are separated by a time interval of not less than $\frac{1}{10}$ second, thus eliminating cases of mere dicrotism. It is usually associated with reduplication of the first sound, and an analogous cardiogram, being therefore central in origin. It is uncertain whether the second wave arises in the ventricle or in the aorta. It is generally met with in cases of left ventricular hypertrophy in association with disease of the aortic valves of the large arteries.[1]

The Pulsus Bigeminus.—By this term we understand a type of regular irregularity in which the pulse waves instead of being equally spaced occur in groups of two, to be followed by a more or less prolonged pause (see Extrasystoles).

"Ventricular bigeminy which occurs in clinical instances of auricular fibrillation either spontaneously or in the wake of digitalis administration, is due to a disturbance of the irregular series of responses to the auricle by ectopic beats arising in the ventricular musculature" (Lewis). It includes practically all instances of accurate coupling. The two beats should bear a constant time relationship to each other (Wenckebach). If the pulse waves occur in groups of three or four, we describe it as a pulsus tri- or quadrigeminus, respectively.

The Water-hammer Pulse.—The Corrigan or water-hammer pulse occurs chiefly and most characteristically in aortic insufficiency. It is quick, collapsing and large in volume. The diastolic pressure is always low (60 to 40 mm. Hg.), and the systolic pressure generally high (180 to 140 mm. Hg.). Diastole is shortened and the height of the secondary wave is in direct proportion to the amount of arterio-sclerosis present, and not to that of the blood-pressure (Lewis).

The Pulsus Paradoxus.—This term is applied to the disappearance or enfeeblement of the pulse during *inspiration.*

It may occur normally:

(1) During deep inspiration with glottis closed—negative intrathoracic pressure (Mueller's experiment). (2) During forced expiration with closed glottis—high intrathoracic pressure (Valsalva's experiment). (3) If the breath is held after a forced inspiration—pressure on the subclavian artery—glottis open. (The muscles of shoulder girdle compress the subclavian artery between the clavicle and the first rib.)

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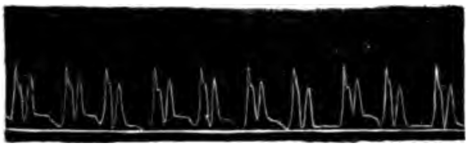
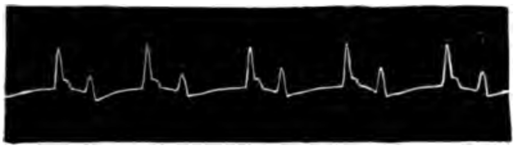
[1] Lewis, T.: *Brit. Med. Jour.*, April 20, 1907.

Normal sphygmogram.

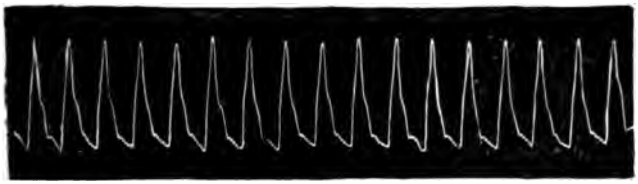
The dicrotic pulse (8th day of pneumonia).



Pulsus bisferiens.

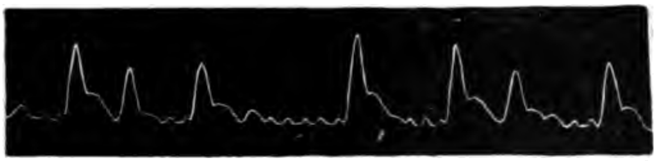


The bigeminal pulse (digitalis effect in auricular fibrillation).



The water-hammer pulse (aortic insufficiency)

Slow high tension pulse (arteriosclerosis).



Pulsus irregularis perpetuus (auricular fibrillation). Irregular both as to time and volume.

Fig. 131.

It may occur pathologically:

(1) At the extremes of life during inspiration. (2) During inspiratory dyspnea—croup—slow inspiration, high negative intrathoracic pressure. (3) In cases of adhesive (mediastino) pericarditis. Respiratory traction resulting from mediastinal adhesions, is brought to bear on some of the large vessels. (It may be a unilateral phenomenon.) If enfeeblement or disappearance occurs during *expiration* it is called *Riegel's pulse.* The **paradoxical pulse has little if any pathologic significance.** The pathological paradoxic pulse is generally assumed to be due to constriction of some of the large vessels by mediastinal adhesions. The paradoxic venous pulse—filling of the veins during inspiration—has a similar genesis.

CHAPTER XIV

RUMENTAL METHODS, BLOOD-PRESSURE ESTIMATION

lse tension or blood-pressure is best estimated by means of a rial sphygmomanometer equipped with a cuff, not less than 12 cm. lth. For children smaller sizes may be employed.

TECHNIC

e Systolic Pressure.—The cuff is applied snugly to the upper arm cured by means of straps or a bandage. The cuff is then connected he manometer and quickly inflated to a point well above the sys-

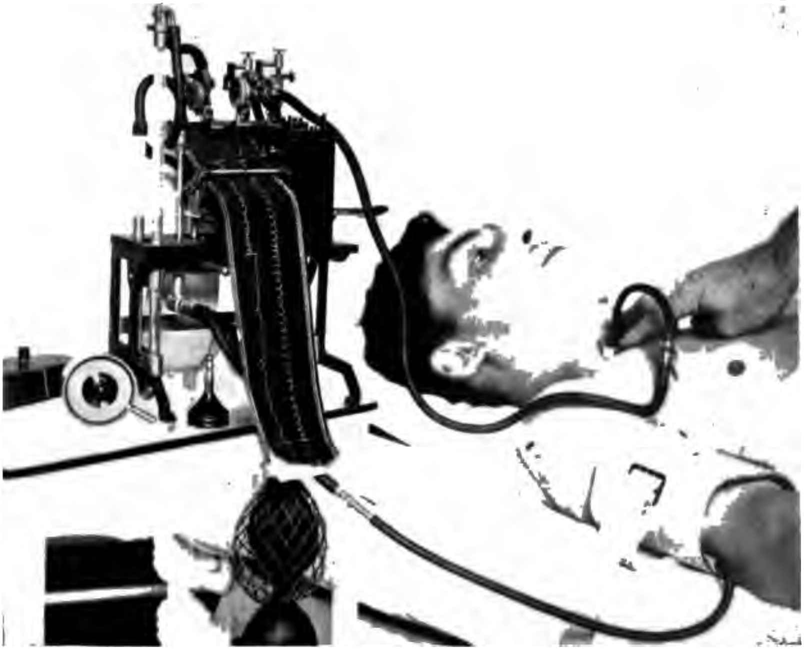


Fig. 132.—The Uskoff sphygmotonograph.

pressure, after which the stop-cock is turned, so as to exclude the from communication with the cuff. A stethoscope is now placed the brachial artery below but not in contact with the cuff, and auscul-n is practised while the pressure is gradually allowed to fall. The lear *thumping or pounding noise indicates* the fact that pressure in rtery is now sufficiently high to force the blood under the constricting and to distend the artery below. The height of the mercurial column at which this acoustic phenomenon occurs indicates *the systolic pressure* in the brachial artery.

The Diastolic Pressure.—The examiner should continue to listen while the mercury falls, and in doing so will notice after a time, that the thumping sound is followed by a "hiss" or murmur which in turn disappears and is replaced by a clear-cut sound similar to that first heard. As the mercury falls still lower this clear-cut *sound suddenly becomes muffled and distant.* This, the beginning of the fourth auscultatory phase, indicates *the diastolic pressure.*

Occasionally it happens that the fourth phase cannot be accurately identified. In such cases the fifth phase (the disappearance of all sound) is chosen as the diastolic criterion. The difference between these two phases rarely exceeds a few millimeters of mercury, but at times they may be separated by 15 mm. or more. The fifth phase is sometimes persistent down to 0 mm. Hg. in *aortic insufficiency.* In these cases the diastolic pressure must be estimated by the fourth phase or not by auscultation at all.

For purposes of comparison, blood-pressure readings should be made (1) in the same posture; (2) on the same arm; (3) in about the same relation to meals. It should be remembered that pain, fear, anxiety, excitement, exercise, etc., may cause marked increase in pressure although in such instances it is the systolic pressure which is chiefly affected. The readings should be made as quickly as is consistent with accuracy. After estimating the pressure the air should be allowed to escape from the cuff and after the lapse of a few minutes a control observation should be made. This will often be found to be lower than the first reading, especially in patients unaccustomed to the procedure, the initial high reading being due to psychic influences. If the cuff remains inflated for too long a time, the resultant venous stasis will *per se* increase the pressure.

NORMAL BLOOD-PRESSURE

In healthy adults the normal systolic pressure is about 130, the diastolic pressure about 80 mm. Hg. During the first month of life the systolic pressure ranges between 60 and 90 mm. Hg. and gradually increases. This increase bears a more constant relation to height and weight than to age or sex. The diastolic pressure in infants is very difficult to estimate owing to the small size and relatively deep situation of the artery.

As age increases pressure gradually tends to rise, generally at the ratio of about 1 mm. Hg. for every two years of life, assuming that a youth of twenty years has a pressure of 120 mm. Hg. *Pressure readings above 160 mm. (systolic) or 100 mm. (diastolic) if constantly present must be considered pathologic at any age.*

THE SIGNIFICANCE OF BLOOD-PRESSURE ABNORMALITIES

Increased blood-pressure is clinically more common and diagnostically far more significant than decreased blood-pressure.

Hypotension.—Occurs chiefly in early life (constitutional or essential hypotension) and is often associated with visceroptosis and sometimes with tuberculous disease. It is also met with after exhausting fevers, in cachexia, as a terminal phase of hypertension, and in shock.

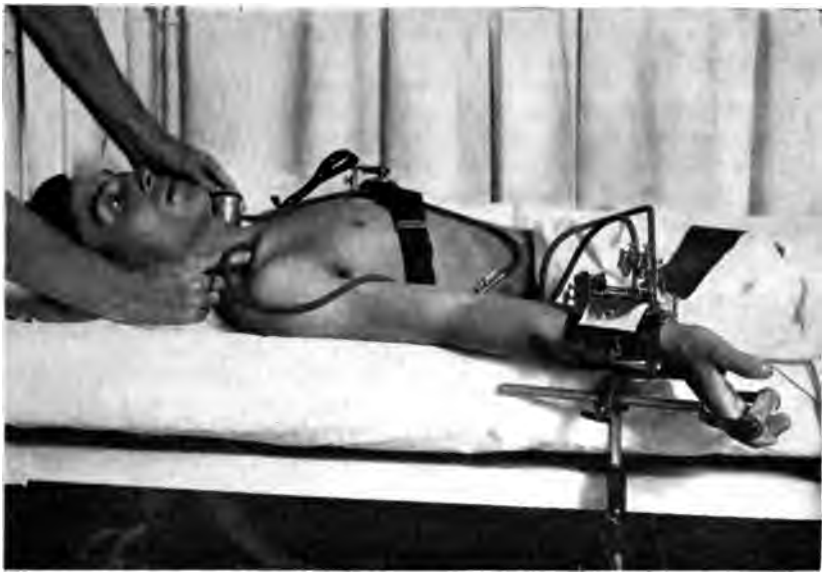


FIG. 133.—The Jacquet cardiosphygmograph. The jugular vein on the right, and the carotid artery on the left as well as the radial pulse are being simultaneously recorded.

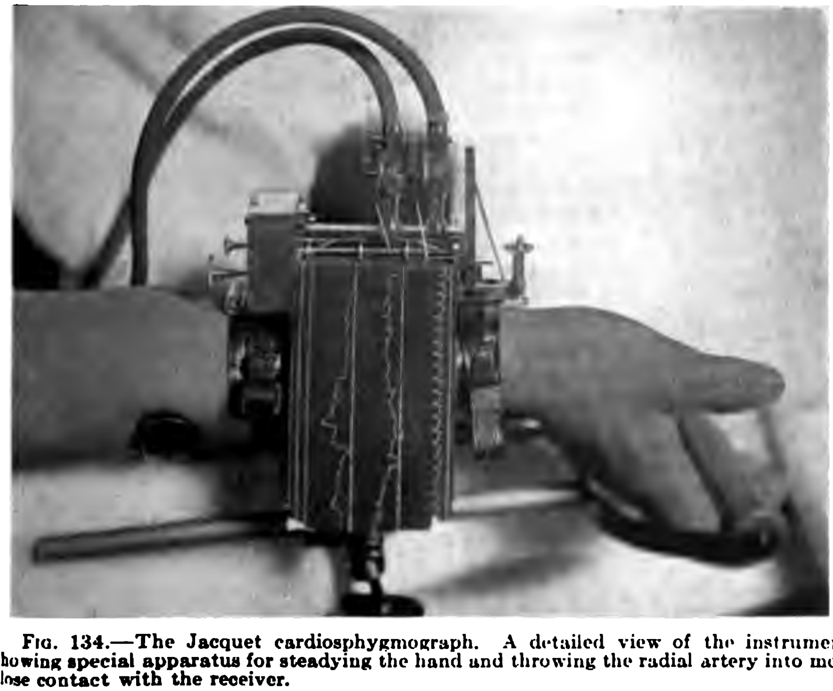


FIG. 134.—The Jacquet cardiosphygmograph. A detailed view of the instrument, showing special apparatus for steadying the hand and throwing the radial artery into more close contact with the receiver.

Hypertension.—Occurs chiefly after middle life and suggests the clinical symptom complex known as Bright's disease—arteriolar disease with cardiac hypertrophy and more or less destruction of renal tissue, terminating in cardio-vascular failure, uremia, apoplexy or angina pectoris.

Hypertension may also occur in plumbism, polycythemia, etc., or it may be temporarily present as the result of anger, fright, excitement or physical exercise. It may occur locally as a result of vascular spasm (*vascular crises*) and cause intermittent claudication, blindness, aphasia, angina pectoris, etc.

VENOUS BLOOD-PRESSURE

The clinical estimation of venous blood-pressure is attended with much more difficulty and much less accuracy than in the case of arterial tension. A rough clinical test may be made by noting the level at which the veins of the hand collapse while the arm is being gradually raised from the level of the hip to above that of the shoulder. Collapse normally should occur at xipho-sternal articulation (Gaertner's test). Venous pressure is estimated in centimeters of H_2O. The normal pressure at the level of the auricles (Louis' angle) ranges between 5 and 10 cm. H_2O. It is increased by exercise, weakness of the right heart, etc., and may reach as high a figure as 30+ cm.[1] An increase in venous pressure generally occurs with a fall of arterial pressure when cardiac compensation fails.

THE VENOUS PULSE

Relatively little knowledge can be derived from a sphygmographic tracing of the arterial pulse alone, but combined, simultaneous records taken from the jugular vein and from the radial or brachial artery are often of extreme importance and are, indeed, the chief method by which the different forms of cardiac arrhythmia are clinically studied. The phlebogram from the jugular vein depicts the activity of the right auricle, the sphygmogram of the brachial artery that of the left ventricle. The cardiogram—the tracing taken from the apex impulse—is often unsatisfactory and clinically of minor importance.

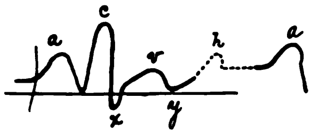


FIG. 135.—Normal phlebogram.

The **normal venous pulse** consists of three, sometimes of four curves.

The "a" wave is a stasis wave due to the contraction of the right auricle. It is presystolic in time. *The "c" wave* is a true venous wave due to contraction of the right ventricle, but in clinical tracings made over the jugular vein this wave is in part due to the impulse transmitted from the neighboring carotid artery. The interval which elapses between the beginning of the "a" and the "c" waves is known as *the conduction time*. It represents the length of time required for the stimulus which causes the heart to contract, to pass from the auricles through the auriculo-ventricular bundle to the ventricles and to produce contractions in these chambers. The time normally required is 0.2 second with a pulse rate of 80 per minute. An increase of the "a–c" time often indicates a lesion in the bundle of His.

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[1] For more detailed information see NORRIS, G. W.: "Blood-pressure, Its Clinical Application," Philadelphia, 1917.

The "v" wave is a stasis wave which occurs while the ventricle is filling with blood, and which disappears suddenly when the auriculo-ventricular valves open. The "v" wave occurs early, is large and prolonged when the right ventricle is overloaded. In cases of tricuspid insufficiency the "c" wave disappears and is supplanted by a large "v" wave. The venous pulse under these circumstances is spoken of as a **"positive venous pulse."**

The "h" wave is not normally present. It is due to a flapping together (upward) of the tricuspid and mitral leaflets, and is protodiastolic in time. It is synchronous with the third heart sound and may occur (1) in slowly beating hearts (because of relatively early diastolic ventricular distention); (2) in aortic insufficiency (because of high intraventricular pressure); (3) in the early stages of mitral stenosis (rapid ventricular filling due to a distended auricle).

SPHYGMOGRAPHS

Numerous instruments have been devised for the recording of arterial and venous pulse waves. The author personally prefers *the Uskoff sphygmotonograph*, which may be obtained either with or without a manometer for estimating blood-pressure. The driving mechanism which propels the smoked paper is equipped with two speeds and very excellent records from the brachial artery and the jugular vein can be made in a very few minutes.

The *Jacquet cardio-sphygmograph* has the advantage of permitting one to make three simultaneous records, but as already stated the cardiogram is of little value in the average case. The respiratory movements can, however, be traced instead of the cardiac impulse. Like all instruments which are attached to the wrist, the adjustment is more troublesome and more time is required to make a tracing.

The Mackenzie polygraph is much used, but in our experience the jugular tracing is not as satisfactory as with the above-mentioned instruments, and a good deal of time is sometimes required for adjustment. The tracings are made with ink pens upon white paper which runs off a spool, so that very long tracings can be made. This is a feature of undoubted utility in some cases.

General Technique.—For the purpose of making *jugular tracings* the patient must be in the recumbent position, with the head as low as possible. A small pillow under the head, however, is sometimes necessary in order to relax the sternomastoid muscle at the origin of which eceiver is placed. When respiratory excursions are deep, it is often ssary for the patient to hold his breath while the phlebogram is being e, since the oscillation of the supraclavicular structures may confuse venous waves. The Uskoff cuff requires no adjustment other than ation to about the level of the diastolic pressure. With the Jacquet trument, however, the receiver must be placed exactly over the *radial ery.* If the course of the artery is outlined upon the skin by means of a blue pencil, this procedure is much facilitated. The patient must be cautioned not to move the hand once the instrument has been adjusted.

The *cardiogram* should be taken over the outermost portion of the apex impulse. In many cases no impulse may be perceptible unless the patient rolls more or less upon his left side. The cardiogram of the right ventricle may often be obtained in or near the epigastrium. In contradistinction to that of the left ventricle it consists of a systolic retraction, not an elevation.

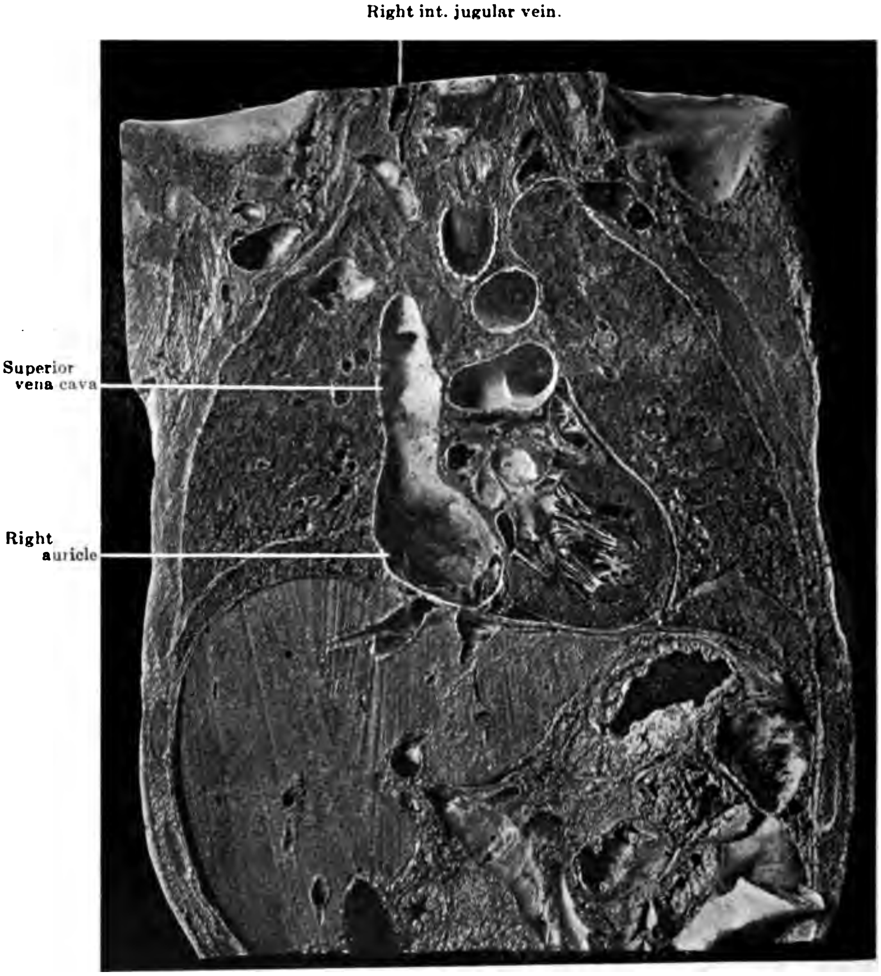

FIG. 136.—The right internal jugular vein empties through the right innominate vein into the superior vena cava. The distance between the jugular bulb over which phlebograms are taken is not far from the right auricle and since no venous valves are interposed, auricular pressure changes promptly produce relatively similar changes in the jugular vein.

The *respirations* may be charted by placing a cup receiver in the episternal notch or by placing the cardiograph attachment in the epigasrium and maintaining it in position by means of an elastic strap. The egistration of the respiratory curve is chiefly important in relation to nus arrhythmia. In most of the instruments in clinical use the time is ecorded in fifths of a second.

It is necessary that either (1) the recording needles be directly in line here the tracing is made, or (2) that any discrepancy which may exist etween their position be shown upon the tracing so that allowance may e made for it in identifying the venous waves. This is shown in Fig. 37 in which the driving mechanism has been stopped near the middle f the tracing and the relative position of the recording needles is shown y the vertical strokes of the lever.

THE INTERPRETATION OF PULSE TRACINGS

First.—The first point requiring attention is the identification of the c" wave in the jugular tracing. This is accomplished by selecting the rave which very slightly precedes the systolic wave in the brachial racing. In doubtful cases the receiver may be moved up higher along he edge of the sternomastoid muscle where a pure carotid tracing can be btained.

Second.—The "a" wave is identified by the fact that it immediately recedes the "c" wave. It appears from 0.15 to 0.2 second before the atter and almost synchronously with the "a" wave of the cardiogram ince there are no venous valves interposed between the right auricle nd the jugular bulb (see Fig. 137).

Third.—The "v" wave follows the "c" wave after a variable interval. t is often separated from the former by a sharp negative depression "x" which is chiefly due to negative auricular pressure due to displacenent of the auriculo-ventricular tissues during ventricular systole.

The "v" wave is sometimes split, the first portion "v" being due to entricular systole and occurring during the latter part of the systolic lateau in the cardiogram. The second portion "v^2" occurs at the end f this time and is due to the downward rebound of the auriculo-entricular tissues to their normal position.

The second negative depression "y" is due to the opening of the uriculo-ventricular valves.

CHAPTER XV

CARDIAC ARRHYTHMIA

Recent additions to our knowledge of cardiac anatomy and physiology have thrown much valuable light upon our conception of heart disease, and have placed cardiac diagnosis and therapy upon a much more accurate scientific basis. As a result of these advances the study of cardiac irregularities has been greatly simplified, and the classification of these conditions has changed from the hopeless jumble which existed a few years ago, to a small and relatively simple grouping.

NORMAL RHYTHM

The stimulus which causes the heart to contract arises in the ***sino-auricular node***, a small collection of specialized right auricular tissue which lies "in the sulcus terminalis just below the fork formed between the superior surface of the auricular appendix and the superior vena cava."

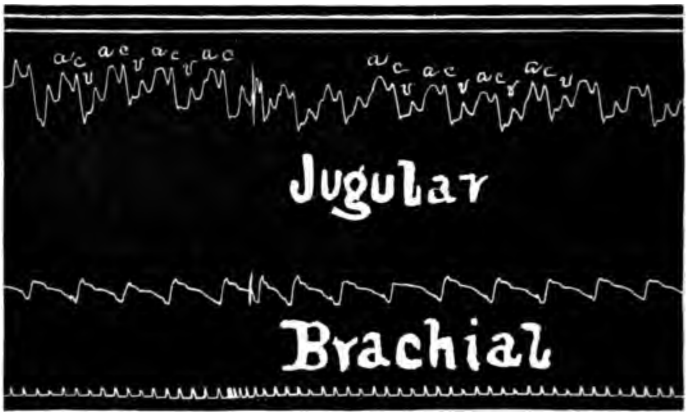

FIG. 137.—*Normal phlebogram.* Blood-pressure *S.* 130, *D.* 78 mm. Hg. Note the regular sequence of the "a," "c," "v" waves bearing a definite time relationship to the brachial arterial waves. The conduction time ("a–c" interval) is normal.

The impulse is conducted from the auricles to the ventricles by means of the *auriculo-ventricular bundle* (His' bundle), a network of specialized tissue which begins in the right posterior margin of the base of the interauricular septum (the auriculo-ventricular or Tawara's node) and narrowing to form a bundle of fibers, leads through the interventricular septum to the ventricles. Before entering these structures it divides into two branches (a right and a left) and again spreads out into a network which is distributed to the papillary muscles and to the ventricular walls, and causes a contraction of these structures.

Normal cardiac contraction is due to the fact that this organ possesses the functions of (1) *stimulus production*, (2) *excitability*, (3) *conductivity*, (4) *contractility*, and perhaps (5) *tonicity*. Further, that these functions are definitely coördinated. The cardiac nerves have apparently both a direct and an indirect modifying and controlling function.

If any portion of the foregoing mechanism fails in its proper, coördinated function either from the standpoint of time, place, or intensity, arrhythmia will result. Thus, for instance, the stimulus may be generated too rapidly, or from abnormal regions (heterogenetic stimuli), as in auricular fibrillation, so that the ventricles cannot keep pace. Or it may be that the auriculo-ventricular bundle is diseased and fails to conduct impulses normally from the sinus node to the ventricles, and heart block results. Again, the excitability of the ventricles may be increased so that they respond to abnormal stimuli and contract before the proper time, producing ventricular extrasystoles; or the ventricular muscle may be deficient in contractile power and contract with irregular force (pulsus alternans).

The term *allorhythmia* (altered rhythm) is applied to cases which show a regular irregularity (*e.g.*, 2 to 1 heart block) and *pararhythmia* to those in which two separate rhythms are simultaneously seen in the same or different heart chambers. Several different types of irregularity may coincidently be present in a given case. Thus extrasystole not infrequently complicates the other forms. Among the pathologic types of arrhythmia, extrasystole and auricular fibrillation are the most common. Auricular fibrillation is next in order of frequency, the third place being filled by pulsus alternans, and the rarest being heart block.

The vast majority of cases of cardiac irregularity will upon analysis be found to conform to one of the following types. The modern classification of heart irregularities, based as it is upon variations of definite physiologic functions of the heart mechanism, has greatly simplified the whole question.

Irregularity of the pulse occurring within the first ten years of life is usually due to sinus arrhythmia. The relative frequency of different arrhythmias from adolescence to old age as seen in general hospital practice is auricular fibrillation 40 per cent., extrasystole 35 per cent., pulsus alternans 15 per cent., paroxysmal tachycardia, sinus arrhythmia, heart block or flutter, 10 per cent. A persistent pulse rate of 35 suggests complete heart block; of 40–50, partial heart block. On the other hand a continual rate of 130 leads one to expect either auricular flutter or a prolonged paroxysm of tachycardia. An irregular pulse of 120 or more usually means auricular fibrillation. Extra-systoles are unusual with a pulse of or over 120. The more persistent the irregularity and the higher the pulse rate, the greater is the likelihood that we are confronted with fibrillation (Lewis).

TACTILE DIAGNOSIS OF THE ARRHYTHMIAS

Pulse Intermission.—If a pulse of regular rhythm is interrupted from time to time by a pause, we are dealing either with (*a*) extra- [illegible] common; the intermission corresponds with a heart impulse [illegible] at the precordium; or (*b*) *heart block*—rare; no impulse or [illegible] the precordium.

[illegible] patient is up and about and has not symptoms of cardiac [illegible] we are almost surely dealing with extrasystoles. If marked [illegible] compensation exist the probabilities are in favor of

"*Coupled Beats.*—If the *ventricular* beats are coupled and the couples are evenly spaced they are the result of one of two mechanisms: 1. for either the alternate beats of the normal rhythm have been replaced by premature contractions—in which case the second beat of the couple is weak and may not reach the wrist—or 2. each third ventricular contraction has been lost and heart block is present. If the *pulse* beats are coupled (*pulsus bigeminus*) a third possibility remains; the pulse pairing may be due to the occurrence of premature heart beats which replace each third rhythmic beat. If such is the case the premature beat will be appreciable at the apex, though it does not reach the wrist."

"*Triple Beating.*—The recognition of the cause proceeds along similar lines. Tripling at the *apex* is due to premature contractions which replace each third rhythmic beat, or to heart-block in which each fourth ventricular contraction has been lost. Tripling of the *pulse* (*pulsus trigeminus*) may be due to a third cause, namely, premature beats replacing each fourth rhythmic beat, the early beat failing to reach the wrist."

"*Halved Pulse Rate.*—When the ventricle beats at twice the pulse rate, the disorder is due to premature contractions in all but the rarest instances. Alternation has been known to occasion halving, the weak alternate beats failing to reach the wrist; but this condition is of great rarity and, when it occurs, is very transient. The two are readily differentiated, for in the first instance the ventricular beats are coupled while in the last they appear regularly."

"When sudden and exact halving of pulse rate is noted and the ventricular rate is halved simultaneously, the disorder is the result of heart-block."

"*A grossly irregular pulse* in which there is hopeless jumbling of stronger pulsations, with quick runs of almost imperceptible beats, and in which the lengths of intervening pauses are constantly varying, is due to auricular fibrillation."

"*A mild grade of irregularity* which persists, which is not related to respiration even when the breathing is deepened and in which no definite sequence of events can be determined, is also due to auricular fibrillation in most cases. A similar irregularity, which shows relations to respiration, is a sinus arrhythmia" (Lewis).[1]

SINUS ARRHYTHMIA

Under normal conditions a deep inspiration tends to accelerate, a deep expiration, to retard the pulse. If this condition becomes exaggerated so that ordinary breathing produces a distinct pulse irregularity, we speak of it as sinus arrhythmia. The name juvenile arrhythmia has also been used on account of the frequency with which it occurs in children. It is essentially a vagus phenomenon, being due to overactivity of the tenth nerve. Sinus arrhythmia may also occur independently of the respiratory act. It occurs characteristically during convalescence from infections, can be abated by full doses of atropin and possesses no pathologic significance. Its recognition becomes important mainly for the purpose of excluding serious forms of arrhythmia during convalescence in diphtheria, etc., or when myocardial disease is suspected. The heart nds remain unaltered during sinus arrhythmia, only diastole being cted.

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[1] Lewis, Thos.: "Clinical Disorders of the Heart Beat." N. Y., 1916. (An excellent, brief and practical handbook.)

HEART BLOCK

This form of arrhythmia is characterized by bradycardia (pulse rate o 10 per minute) and by the fact that venous pulsations in the jugular ns are visible more frequently than ventricular contractions over the cordium. When the bradycardia is associated with periods of asystole,

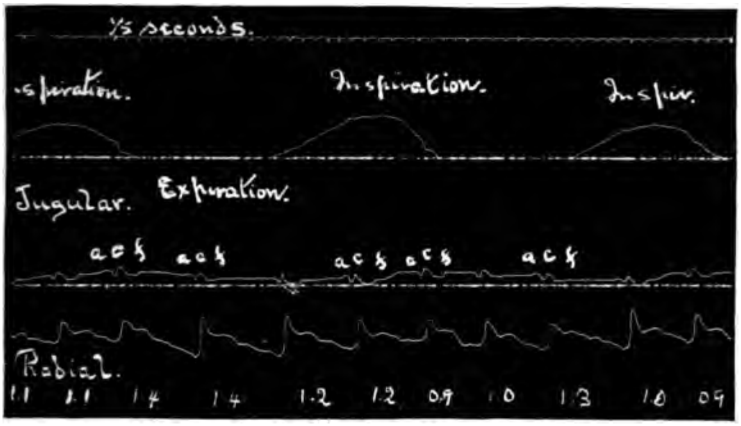
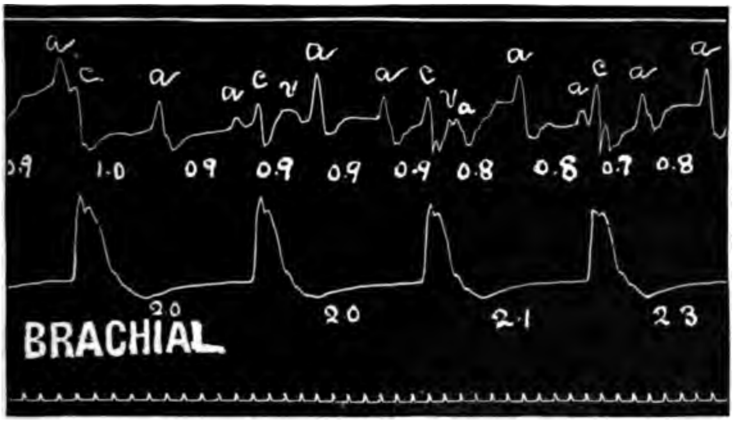


FIG. 138a.—*Sinus arrhythmia*, showing pulse acceleration during inspiration.



138b.—*Auriculo-ventricular heart block*, with the auricles beating twice as fast as the ventricles, the rate of the latter being 30 per minute.

ncopal attacks or convulsions, the condition is known as the *Adams-okes syndrome.* The phlebogram shows from two to four "a" waves r each "c" wave. The lesion generally lies in the auriculo-ventricular undle and is frequently syphilitic in origin. More or less destruction the bundle "blocks" the stimuli between the sinus node and the venicle. When the block is complete the ventricle initiates its own rhythm (about 30 per minute). If the block is incomplete every second, third, or fourth sinus impulse produces a ventricular contraction and the condition is spoken of as a 2 to 1, 3 to 1, or 4 to 1, block. Incomplete block often shows a prolongation of the "a–c" interval. The conductivity time is also frequently increased in rheumatic valvular lesions. The abortive auricular systoles can sometimes be heard over the precordium as ill-defined muffled heart sounds. Heart block is always serious and frequently a fatal condition. Some seven cases of the Adams-Stokes syndrome have, however, been known to recover. Temporary block (especially in the milder forms of prolongation of the "a–c" interval, or an occasional dropped beat) may be caused by full doses of digitalis and similar remedies. It also may occur in several of the acute infections, especially rheumatic fever, under which circumstances it is due to acute myocardial inflammation involving the conductive system.

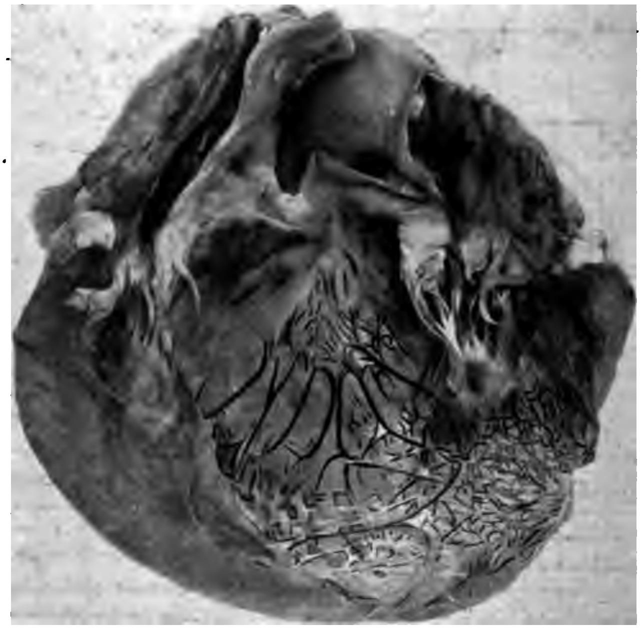


Fig. 139.—Auriculo-ventricular bundle in a human heart, injected with India ink. (*Courtesy of Dr. F. T. Fulton.*)

EXTRASYSTOLE

Under normal conditions ventricular contractions are due to stimuli originating in the sinus node and transmitted through the auricle and the bundle of His to the ventricle. The contractions occur at regularly spaced intervals. Under abnormal conditions additional effective stimuli may arise at other portions of the heart (auricle, ventricle, bundle). Such stimuli interfere with the regular rhythm by producing premature contractions—extrasystoles. Such abnormal stimuli are most apt to occur when the cardiac rate is slow. The muscle becomes impatient in waiting for its signal and contracts without orders. The sound produced by an extrasystole is less intense than the normal sound, and may even be inaudible.

Ventricular extrasystoles are characterized by the fact that they are followed by compensatory pauses, so that the time occupied by the systole and the extrasystole is equal to that of two normal contractions. Also by the fact that the extrasystole occurs early in diastole and produces either a weak or a missed beat at the wrist while the succeeding one is forcible, so that the patient is often conscious of it. The compensatory pause is due to the fact that the next normal stimulus reaches the ventricle so soon after the premature contraction that the latter is still in the refractory phase. Ventricular extrasystoles may occur without interfering with any of the normal stimuli, in which case the pause after the premature contraction is shorter than normal, and an "interpolated" extra-

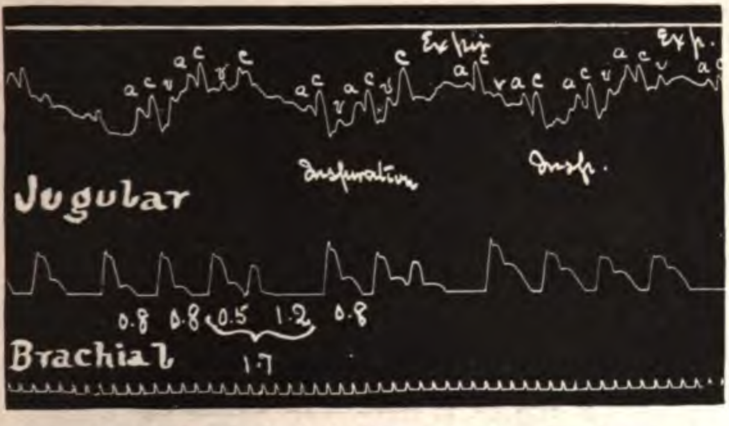


FIG. 140.—*Extrasystoles,* ventricular in origin, followed by compensatory pauses.

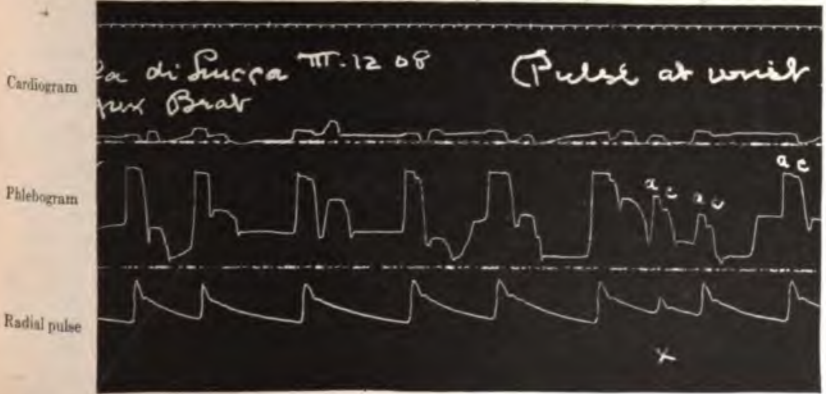


FIG. 141.—*Coupled beats.* The coupling is visible in the jugular and apex tracings, only occasionally in the radial. The case was one of auricular fibrillation under full digitalization. The radial pulse rate is about one-half of that of the heart. An extrasystole occurred at "x."

systole occurs. Inspection of the phlebogram shows the absence of an "a" wave to correspond with the extra "c" wave; and auscultation of the precordium often demonstrates the absence of a second sound to correspond with the ventricular contraction.

Auricular extrasystoles are due to stimuli arising at some point of the auricle other than at the sinus node. The phlebogram shows an "a" wave preceding each extrasystole of the ventricle. The compensatory pause is generally absent, but may be almost or even wholly complete. Auricular extrasystoles may be induced or abated by digitalis.

The significance of extrasystoles is very variable. They may be due to many direct or reflex causes. Thus the excessive use of tobacco may produce them, as may increased blood-pressure, especially with a weakened myocardium.

PAROXYSMAL TACHYCARDIA

This term is applied to a distinct pathologic entity due to a disturbance of the intrinsic cardiac mechanism. It should not be applied to simple tachycardia due to extrinsic causes such as exophthalmic goitre. Clinically this condition is characterized by sudden attacks of rapid heart action in which the previous cardiac rate is approximately doubled, trebled or quadrupled. Ordinary tachycardia rarely produces a pulse rate over 140 per minute, in paroxysmal tachycardia the rate ranges between 140 and 280 per minute. These attacks may be precipitated by diverse causes such as excitement, change of posture, flatulence, exercise, etc.; and end after a variable duration as abruptly as they began. They are frequently associated with subjective sensations of palpitation, oppression, fullness of the neck, dyspnea, vertigo, etc., the intensity of which often depends upon the integrity of the heart muscle.

Venous and electrocardiographic studies taken at the beginning and end of the paroxysms have shown that the cardiac rhythm may be variable. Two types of this phenomenon have been described:

1. The type in which the *auricular type* of venous pulse persists, in which the end of the attack may terminate with normal coördination or with a temporary auriculo-ventricular block.
2. A second rarer *ventricular type* has also been observed in which the paroxysms are characterized by the entire absence of auricular waves, which do, however, reappear at the end of the attack, without any evidences of auriculo-ventricular block.

The etiology of paroxysmal tachycardia is obscure. Numerous explanations such as the interpolation of extrasystoles, abnormal stimulus production in the Purkinje cells of the auriculo-ventricular bundle, etc., have been suggested. Practically the condition may be regarded as due to an increased irritability of the myocardium in which diverse reflex stimuli may precipitate a paroxysm.

The effects upon the circulation are those of a shortened diastole with insufficient ventricular filling, as the result of which arterial pressure falls, venous pressure rises and variable degrees of local or general circulatory stasis may occur.

AURICULAR FLUTTER

Auricular flutter is a not uncommon form of arrhythmia which occurs chiefly in elderly subjects. It is characterized by an extremely rapid *auricular action,* the rate being from 200 to 400 per minute (usually above 300). When established it generally lasts for months or years, but paroxysms may be shorter. It is due to pathologic or heterogenetic impulses which probably have an ectopic auricular origin. It is not under nerve control. The rate is wonderfully constant as a rule, and is uninfluenced by position, exercise or stimulation. The *ventricular rate* is generally one-half the auricular rate, but any degree of block may be present. The ventricle may beat regularly if the degree of partial block is constant, or irregularly, if the degree of block varies (*e.g.*, 3 to 2, 2 to 1, etc.). Auricular flutter is closely related to paroxysmal tachycardia. It is much influenced by digitalis and vagal compression. Digitalis may change auricular flutter to fibrillation and the latter to a normal rhythm or it may have no demonstrable effect. The arterial tracings may be regular or irregular, fast or slow, and may suggest fibrillation or extrasystoles. The venous curves are generally obscure on account of auricular weakness, but when pauses are long "a" waves may be distinct (T. Lewis).[1]

AURICULAR FIBRILLATION

This common form of arrhythmia occurs in *serious organic lesions of the heart muscle.* It can be produced experimentally by electric overstimulation of the auricle and may be regarded as manifestation of auricular exhaustion. It occurs mainly in rheumatic endocardial lesions, especially in long standing mitral stenosis with right auricular dilatation, but is also seen in arterio-sclerotic lesions. It is identical with what was formerly described as "*pulsus irregularis perpetuus,*" "nodal rhythm" and "delirium cordis." It is *characterized by* an absolutely irregular arterial pulse. The pulse waves are irregular both as to force and time, and many ventricular contractions fail to produce a peripheral pulse, hence we find a *pulse deficit* in the radial and other peripheral arteries. The phlebogram shows the absence of "a" waves and the presence of a large "v" wave, which can also often be seen as a systolic venous pulsation in the jugular veins. The foregoing characteristics are due to the fact that the ventricle instead of receiving the normal stimuli from the sinus node transmitted through the auriculo-ventricular bundle at regular intervals, and with definite intermissions, is overwhelmed by stimuli received so frequently that diastolic filling and rest are impossible. Digitalis in sufficient dosage produces a specific reaction which is attended by excellent clinical results. This is accomplished by lowering the conductivity of the auricular-ventricular bundle, so that fewer stimuli reach the ventricle. Excessive *digitalization* often results in the production of a *bigeminal pulse* (p. 161). A rare form of auricular fibrillation exists in which attacks occur in paroxysms lasting a few hours, days or weeks. They may be brought on by emotion or physical strain, and may cause severe symptoms of cardiac distress and palpitation. By *palpitation* we understand rapid or tumultuous heart action of which the patient is subjectively conscious. Auricular fibrillation is a serious form of arrhythmia which once it has set in generally persists, although life under restricted conditions may last several years.

Blood-pressure in auricular fibrillation often cannot be estimated by the usual methods, owing to the variable size and force of succeeding pulse waves. This difficulty may be overcome by the method suggested by James and Hart:

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[1] Lewis: "Heart," IV, 1912, 171.

"The apex and radial impulses are counted for one minute, then a blood-pressure cuff is applied to the arm, and the pressure raised until the radial pulse is completely obliterated; the pressure is then lowered 10 mm. and held at this point for one minute while the radial pulse is counted; the pressure is again lowered 10 mm. and a second radial count is made; this count is repeated at intervals of 10 mm. lowered pressure until the cuff pressure is insufficient to cut off any of the radial waves (between each estimation the pressure on the arm should be lowered to 0). From the figures thus obtained the average systolic blood-pressure is calculated by multiplying the number of radial beats by the pressures under which they came through, adding together these products and dividing their sum by the number of apex beats per minute. The resulting figure is what we have called the 'average systolic blood-pressure.'"

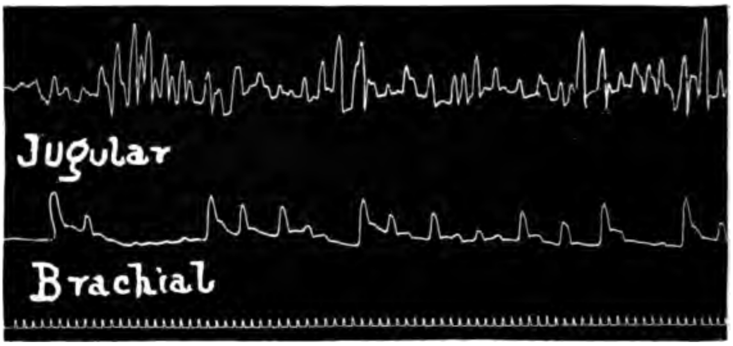

FIG. 142.—*Auricular fibrillation* showing absolute irregularity of the brachial pulse with a deficit, as contrasted with the phlebogram. The latter shows an absence of "a" waves and is of the positive or ventricular type. From a case of broken compensation occurring in rheumatic mitral and tricuspid disease.

The following observation made on a patient will indicate the method of computation:

B. S., April 29, 1910. Apex, 131; radial, 101; deficit, 30.

Brachial pressure		Radial count
100 mm	0	
90 mm	13	13 × 90 = 1,170
80 mm	47 − 13 =	34 × 80 = 2,720
70 mm	75 − 47 =	28 × 70 = 1,960
60 mm	82 − 75 =	7 × 60 = 420
50 mm	101 − 82 =	19 × 50 = 950
	Apex =	131) 7,220
Average systolic blood-pressure		55+

PULSUS ALTERNANS

Pulsus alternans, the third in order of frequency and prognostically one of the most grave forms of cardiac arrhythmia, is characterized by the alternate appearance of large and small arterial pulse waves. The time interval separating the large from the small beats is generally greater than that which separates the small waves from the large ones. This form of arrhythmia, which can be produced experimentally after ligation of a coronary artery if the heart rate is much increased, is due to failure of ventricular contractility and may occur (1) in a normal heart beating at an excessive rate, or (2) in a degenerated myocardium contracting at a normal rate. The rate being too rapid for the myocardium, only a partial systole occurs, since the ventricle is insufficiently rested, but in the weaker systole less energy is consumed and hence the next contraction will be normal in force. According to another explanation, a part of the ventricle altogether fails to contract, remaining in a refractory state and thus causing a weak contraction. Whether this is due to a disturbance of irritability, conductivity, or contractility is still an open question. The last-mentioned function seems to be most commonly at fault. Gravier, who has published an elaborate monograph on the subject, speaks of pulsus alternans as "a disorder of the refractory phase."[1]

The alternation of force so apparent at the wrist is rarely demonstrable at the cardiac apex and the relationship has actually been found reversed—a strong systole being associated with a small radial pulse and *vice versa*. The explanation of this phenomenon perhaps lies in the fact stated by Hering that that part of the cardiac musculature which forms the cardiac apex is not the part which propels the ventricular blood into the aorta.

Lewis has noted a similar divergence in force between the ventricle and the carotid, which he attributes to alternating auricular force. This corresponds with Volhard's tracings which showed an alternation of the "a" waves in the phlebogram, the large jugular wave being followed by the small radial pulse.

"Temporary exhaustion of contractility often occurs with the production of a typical pulsus alternans from an overtaxation of the cardiac musculature, the result of a too rapid rate, such as may occur in paroxysmal tachycardia, auricular flutter, auricular fibrillation, or the long-continued tachycardia which accompanies Graves' disease. Experimentally it may be produced in healthy hearts by electrical stimulation, the injection into the blood stream of digitalis, antiarin, aconitin, and hemolytic serum. It is most frequently encountered, however, when the heart is beating within its normal rate, and when it denotes a grave pathological condition of the myocardium, with failing contractility" (Gordinier).

Pulsus alternans is met with in cases of *nephritis* and *myocarditis*, generally during *failing compensation*. It may be complicated by extrasystoles.

Pulsus alternans must be differentiated from regular bigeminus due to extrasystoles. The latter are to be distinguished from true alternans by the long diastolic time interval, or by the long compensatory pause following the small extrasystolic beats, whereas in true pulsus alternans the longer period follows the large beat, or else the contractions ar [illegible] ually spaced (Gordinier).

[illegible] *phlebogram* in pulsus alternans shows no abnormal waves, but [illegible] interval may be prolonged. Alternation in the intensity, [illegible] ch of the heart sounds is rarely demonstrable. A markedly [illegible] ay on superficial inspection simulate alternation, but this.

[illegible] VIER, L.: "L'Alternance du Coeur," Paris, 1914.

source of error may readily be excluded by counting the pulse over the precordium. Alternation is frequently overlooked if sphygmographic tracings are not made. The sphygmograph is also useful in demonstrating *latent cases* of alternation. It also enhances one's tactile perception of this arrhythmia. "The cuff is inflated to the point at which the pulse rate becomes halved. If the cuff pressure is allowed to fall somewhat lower all the beats will come through, but the alternation in their size becomes more noticeable than when the arterial lumen is uncompromised."[1]

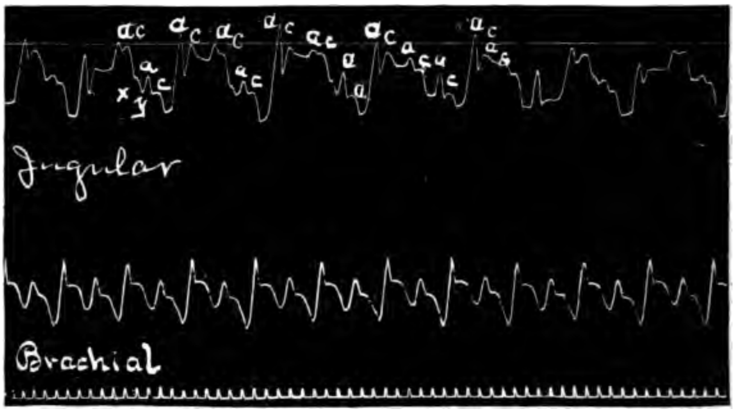

FIG. 143.—*Pulsus Alternans* from a case of cardio-vascular sclerosis with angina pectoris. This arrhythmia was apparently precipitated by digitalis. It was most marked when blood-pressure was at its highest (S. 190, D. 140 mm. Hg.). Death three months later. Note the alternate large and small brachial waves occurring at regular intervals.

THE EFFORT SYNDROME

("Disordered Action of the Heart;" "D. A. H.;" Neurocirculatory Asthenia; Soldier's Heart; The Irritable Heart of Soldiers)

The syndrome upon which these various names have been bestowed, was first described by Hartshorne and by DaCosta during our Civil War. It has been much in evidence in the "Great War" just terminated in which it played a conspicuous part, as a cause of physical disability. Much has been written on the subject which has been carefully investigated by many able men, notably Thomas Lewis,[2] although as yet its exact etiology and pathology still remains unsolved.

Symptoms.—The "effort syndrome" is characterized by dyspnea on exertion, precordial pain, undue exhaustion, vertigo and syncope, palpitation, headache, lassitude, vasomotor disturbance such as coldness, cyanosis, sweating of the extremities, irritability of temper, flushing of the skin, tremor, inability to concentrate mentally. Cutaneous hyperalgesia is frequently present.

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[1] NORRIS, G. W.: "Blood-pressure, Its Clinical Applications," Philadelphia, 1917, p. 250.

[2] Report upon soldiers returned as cases of "Disordered Action of the Heart" (D. A. H.) or "Valvular Diseases of the Heart" (V. D. H.). *Med. Research Comm.*, London, 1914.

The respirations which are rapid (20–60 per min.) and shallow (250–350 cc.) increase greatly on exertion as regards rate, but not in regard to depth. This as has been noted is characteristic of anoxemia as contrasted with an increased carbon dioxide tension of the blood, and these patients while breathing oxygen are able to carry on physical exertion, which, while breathing atmospheric air are quite beyond their powers. Oxygen relieves their subjective symptoms. It is not yet known, however, whether the anoxemia results fron an impaired circulation or from other factors which prevent adequate pulmonary ventilation.[1]

Physical Signs.—Increased *pulse rate* is very common, but lability of the pulse especially in response of emotion or exercise is most characteristic. The systolic *blood pressure* shows an equally exaggerated response of mental or physical effort. The *apex beat* is diffuse, forcible and jerky, is often accompanied by a systolic thrill and accentuation of the heart sounds. The *heart rhythm* is often irregular—intermittent or unduly affected by respiration. *Tremor* of the hands, *vasomotor ataxia*—excessive coldness, blueness, wetness of the palms and soles, increased axillary sudation, slight degrees of fever and tachycardia especially after exertion are commonly noted, as is also capillary leukocytosis.

It is needless to say not every one of the symptoms and signs just enumerated will be present in every case. Dyspnea, pain, undue exhaustion and giddiness are however practically constant features. It will be noted that virtually all the features above referred to are identical with those which might be encountered in a normal individual if the human machine had been driven to the point of exhaustion. In the effort syndrome they occur after the most trivial physical exercise or mental emotion. Practically speaking the Effort Syndrome may be regarded as the response of an individual who is unfit—from a physical, psychic or nervous standpoint to withstand unwonted calls for the expenditure of energy. It is encountered not only in the trenches but also in the training camps hundreds, and even thousands of miles from the firing line.

Diagnosis.—The Effort Syndrome may be closely simulated by (1) hyperthyroidism; (2) mitral obstruction, and (3) gas poisoning. In the early stages a differential diagnosis may indeed be difficult, and thus far neither the history, the employment of the sphygmomanometer, the electrocardiograph or other instruments of precision have added much to our diagnostic ability.

In early *mitral stenosis* exercise sometimes tends to increase the thrill, the murmur and the accentuation of the second sound. In case of the Effort Syndrome the reverse is usually the case. In *hyperthyroidism* the tremor is fine, diarrhea is common, and constant tachycardia the rule. In the Effort Syndrome the tremor is coarse, while the rapid pulse and dyspnea disappear during sleep and rest.

Cases of *gas poisoning* are to be differentiated by the absence of "neurotic" features, for it is to be remembered that the Effort Syndrome presents many parallels with an anxiety neurosis. It must not be forgotten, however, that a man who has been gassed may develop the Effort Syndrome. As Cohn[2] has pointed out surgical cases do not develop the Effort Syndrome with anything like the frequency that medical cases do, especially during convalescence. The leisure to reflect upon the invisible, intangible and to many, mysterious factor, in gas poisoning perhaps plays an important rôle. No one who has seen large numbers of Effort Syndrome cases can fail to be impressed with the importance from an etiologic standpoint of the subconscious mind.

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[1] HALDANE, MEAKINS and PRIESTLEY: "The Effects of Shallow Breathing." *J. Physiol.*, lii, 1919, 433.

[2] COHN, A. E.: "The Effort Syndrome," *War Medicine* ii, Dec., 1918, 757.

THE ESTIMATION OF VASOMOTOR EFFICIENCY

Many tests based upon sphygmographic or sphygmanometric observations for the determination of cardio-vascular efficiency have been devised. Thus far no one is free from criticism, or entirely satisfactory, but the following test which is simple and can be briefly completed, often sheds a useful light upon doubtful cases. It must be remembered, however, that it simply aims to show functional efficiency, not structural integrity.

Crampton's Test of Vasomotor Efficiency.—In rising from the recumbent to the erect posture blood-pressure tends to fall as a result of gravity. Unless this tendency were automatically regulated, syncope would occur as a result of cerebral anemia. In a normal vigorous man, however, such a change of position causes a rise of blood-pressure amounting to 8 or 10 mm. Hg. This increase may result from increased vasomotor tone or from increased cardiac work, or as a result of both factors. Bearing these facts in mind Crampton[1] has devised the following table to test vasomotor efficiency through the observation of pulse rate and blood-pressure responses to postural change. The tables while setting what is perhaps a high normal have shown that vasomotor tone in the same individual varies greatly as a result of mental or physical fatigue, infectious processes, etc. It may be used with especial edification in the study of essential hypotension cases.

The Technique.—The cuff of the sphygmomanometer is adjusted over the brachial artery and the patient is placed on a comfortable couch with a low pillow. The heart rate is counted by quarter-minutes and a gradually decreasing rate is usually observed. Counting should continue until two successive quarter-minutes are the same, this is multiplied by 4 and recorded. The systolic pressure is then taken preferably by auscultation. The patient stands, the heart rate is counted as before until it reaches the "standing normal," when it is recorded, and the blood-pressure is then taken. The differences are calculated and reference is made to the scale.

"This scale provides a convenient and intelligible method of recording and reporting cases and permits a numerical statement of the function in question. Its 100 mark indicates a perfectly efficient working of the vasomotor system under test, the zero is approximately the point where the average person is unable to maintain the erect posture."

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[1] Crampton, C. W.: "The Blood Ptosis Test and Its Use in Experimental Work in Hygiene," *Proceed. Soc. Exp. Biol. & Med.*, 1915, xii, 119.

PERCENTAGE SCALE

Vasomotor Tone

Ieart-rate increase	Blood-pressure										
	Increase					Decrease					
	+ 10	+ 8	+ 6	+ 4	+ 2	0	− 2	− 4	− 6	− 8	− 10
0 to 4	100	95	90	85	80	75	70	65	60	55	50
5 to 8	95	90	85	80	75	70	65	60	55	50	45
9 to 12	90	85	80	75	70	65	60	55	50	45	40
13 to 16	85	80	75	70	65	60	55	50	45	40	35
17 to 20	80	75	70	65	60	55	50	45	40	35	30
21 to 24	75	70	65	60	55	50	45	40	35	30	25
25 to 28	70	65	60	55	50	45	40	35	30	25	20
29 to 32	65	60	55	50	45	40	35	30	25	20	15
33 to 36	60	55	50	45	40	35	30	25	20	15	10
37 to 40	55	50	45	40	35	30	25	20	15	10	5
41 to 44	50	45	40	35	30	25	20	15	10	5	0

Note.—In case of increase in pressure higher than +10 add 3 per cent. to the -10 column for each 2 mm. in excess of 10.

CHAPTER XVI

THE ELECTROCARDIOGRAPH

By Edward B. Krumbhaar, Ph. D., M. D.

Among the instruments of precision recently become available for clinical purposes is the electrocardiograph, adapted from Einthoven's string galvanometer. In the short space of time elapsed since its invention in 1903, it has not only already proved its clinical value as the most accurate analyzer of cardiac arrhythmias, but bids fair to give further information about the cardiac muscle that is obtainable in no other way. In the hands of Lewis, Rothberger and Winterberg, Einthoven and others, it has materially aided in increasing our knowledge of the mechanism of the heart beat, both in health and disease.

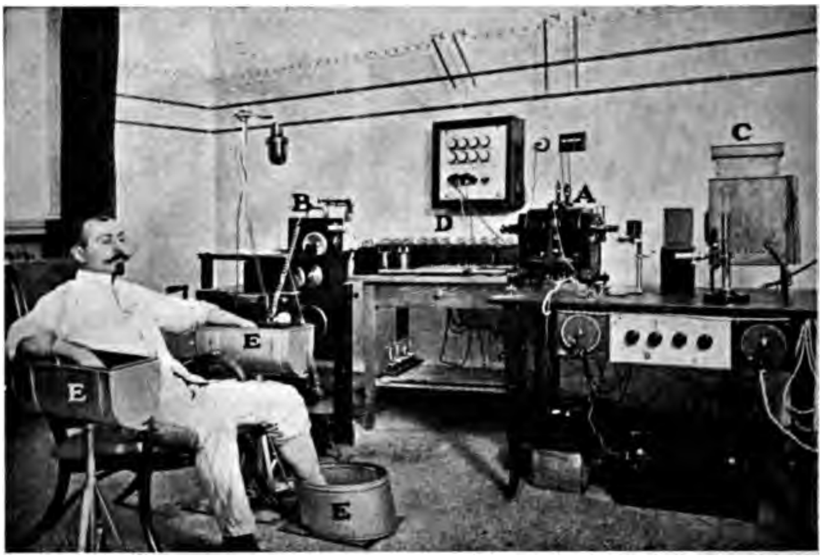

Fig. 144.—An electrocardiograph station. *A*, string galvanometer; *B*, registering apparatus (camera); *C*, electric light; *D*, rheostat; *E*, leads from which current is taken. (*After Hoffman.*)

The Principle.—The string galvanometer depends on the principle that a conducting string or wire lying in a strong magnetic field, if sufficiently sensitive, will move vertically to the lines of force when an electric current passes through it. As every contracting muscle initiates such a current (the part at which the contraction starts becoming electrically negative to the rest of the muscle), the contractions of the various chambers of the heart cause a series of such deflections. These minute currents adiating through the body, if the extremities of the patient are connected ith the galvanometer by suitable non-polarizable electrodes, may be peretuated as a graphic record by photographing the shadow cast by the ibrating string on a moving sensitive film or plate. The galvanometer nd registering apparatus are so arranged that a contraction starting nearer he base of the heart and proceeding toward the apex will cause a deflecion of the vertical string to the left, or upward on standardized records. The field of the electrocardiograph and its limitations will be best comprehended, if it is remembered that it merely registers all changes of electrical otential occurring between the two electrodes in use. Voluntary muscle remors or contractions, when present, are consequently recorded as well as the heart beat.

The three leads (places for attachment of electrodes), adopted by Einthoven as best exhibiting the electrical changes caused by the heart beat, are as follows: lead I, right arm to left arm (horizontal); lead II, ight arm to left leg (long axis of the heart); and lead III, left arm to left eg (vertical). These leads have now come into general use, and should always be taken in routine examinations. Leads may be taken in special cases, however, from any part of the body. A time marker, registering ifths or twenty-fifths of seconds, allows an accurate computation of the various time intervals.

The Normal Electrocardiogram.—The normal mammalian electrocardiogram consists of a single summit or peak ("P" wave), due to contraction of the auricles, and two to four deviations ("Q," "R," "S," "T"), due to contraction of the ventricles. Of these four, "R" and "T" are the most constant, and "Q" and "S" are of little importance n the normal record. Though no two normal hearts give exactly similar records, the individual peculiarities are retained with remarkable constancy as long as the heart stays normal. Although the factors that cause the individual deviations are not yet clearly understood, certain explanations are generally accepted. Thus the monophasic "P" summit is unquestionably due to the impulse starting at the sinus node and spreading through both auricles toward the ventricle. This is followed by an inactive period, normally of 0.12 to 0.17 seconds, called the "P-R" nterval, which corresponds to the "A-C" interval of polygraphic tracngs. Most of this delay is caused by the passage of the impulse through Tawara's node and the bundle of His. The path followed by the contraction stimulus on entering the ventricle is a very complex one, not lending itself to simple representation by such deviations. The "R" deflection was formerly considered as the evidence of the basifugal stimulus conduction in the ventricle. In those cases where it was preceded by "Q," it was assumed that the impulse has first been distributed to tissues near the apex and that the predominating influence for a short period was basipetal (Einthoven). Later experimental and clinical investigations by Thomas Lewis, Buchanan, and de Boer indicate that the form of the "Q," "R," "S" group of the ventricular complex is the result of the algebraic summation of the opposing influences of the right and left ventricle, in other words, a bigram due to the superposition of dextrogram on levogram. From this point of view "Q" in lead I ("Q_1") of the human electrocardiogram is a left ventricular effect, in lead II and III ("Q_2" and "Q_3") a right ventricular effect. Clinical confirmation of this conception is found in the prominent "Q_2" and "Q_3" of infants' electrocardiograms

(Krumbhaar and Jenks). "R_2", "R_3" and "S_1" are due to spread of activity in the left ventricle; "R_1", "S_2" and "S_3" to spread of activity in the right ventricle. The normal limits of the QRS deflection time are between 0.06 and 0.10 seconds. In the isoelectric period preceding "T," a balance is maintained by the whole mass of the heart being in a state of contraction. The final upward deflection, "T," is still taken by some authorities to be an indication of the driving force of the heart, represented by the circular muscle bands at the roots of the great arteries; to others however, the upward T indicates that the impulse to the right ventricle has persisted longer than that to the left. The "U" wave, which is occasionally seen in both normal and pathological records, is unimportant and but little understood. It is taken by Hering to be

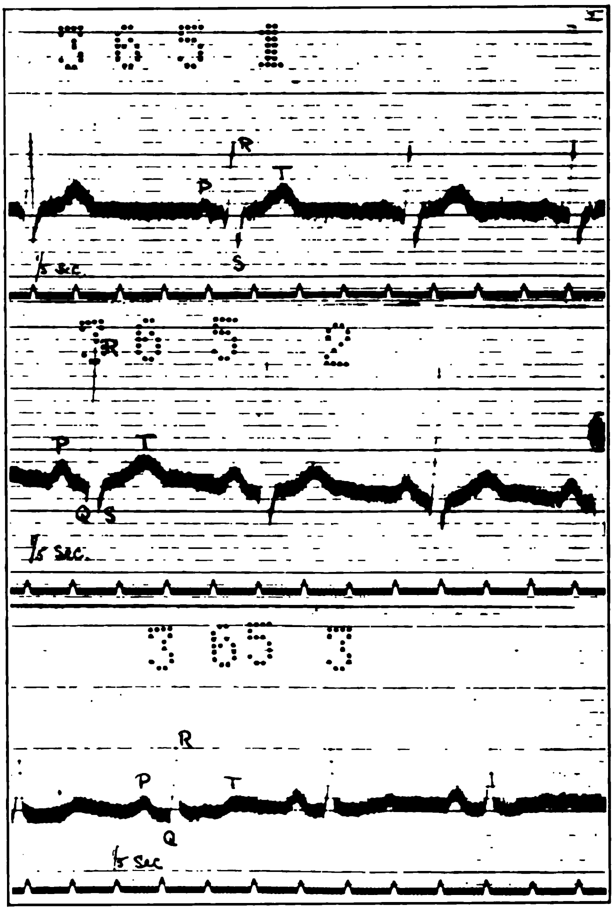

FIG. 145.—Normal electrocardiogram. Taken from the three customary leads. Lead I, right arm to left arm; lead II, right arm to left foot; lead III, left arm to left foot. Note that R_2 is the largest, and that T_3 is small and slightly diphapic. In this, as in all illustrations, 1 cm., deflection of string (*i.e.*, five of the small ruled lines) represents a change in potential of one millivolt. Time record, ⅕ second.

due to changes in electrical potential in the great vessels, *i.e.*, an angiogram. It should be remembered that neither the amplitude nor the duration of the electrical variations are criteria of the force or duration of the contraction of the various heart chambers.

Abnormal Forms of Complexes.—Many variations from what is accepted as the standard form of the electrocardiogram may result from a normal action of the heart, or may have a significance that is as yet but little or not at all understood. If, however, repeated records from a diseased heart show a constant approach to or recession from the standard form, it is very probable that that heart is either improving or getting worse as the case may be. Thus I have seen a notch in an R wave, so deep that it resembled a thin letter M, gradually diminish as the condition of the heart improved, until after recovery a normal R wave was found. Notches may occur on either the up or down stroke of the R or S wave, and in single records little significance can be attached to them. If permanent, they may indicate pathologic changes in the myocardium; if temporary, a transient or potential defect in the intraventricular conducting system. Prolonged deflection time of the QRS group will be considered later under the head of Intraventricular block.

The P wave may be changed in various ways in any of the leads but most frequently in Lead III. It may be either notched, broadened, diphasic or inverted in one or more leads, and either under or independent of vagus control. In the former case, it is probably due to a migration of the pacemaker from the sinus node (Wilson), and in the latter possibly to anomalous position of the sinus node, extra-sinus location of the pacemaker, a change of the heart's position or alterations in muscle balance.

The T wave is frequently diphasic or inverted, and especially in Lead III, where such variations have little significance. In the other leads they are taken by some authors to indicate a bad prognosis; but Cohn has shown that inversion of the T wave can constantly be produced if sufficiently large doses of digitalis are administered. In my own series of cases, negative T waves in Leads I and II in the absence of digitalis medication have usually occurred in very grave cases, and the development of negative T waves in these leads has coincided with an aggravation of the patient's condition. The direction of the T wave is thought by some to depend on the relative duration of stimulus activity in the two ventricles (inverted T due to unusually long duration in the left ventricle). A diphasic T is hard to explain under this conception, unless one assumes a transient preponderating influence of that ventricle which is the first to finish. In some clinical cases, and more often under experimental conditions, the iso-electric line preceding the T wave, instead of being on the usual level, is slightly lower. According to the above conception, this would indicate a preponderance of left ventricular activity continued during most of systole. Its significance is not yet understood.

Preponderating Ventricular Hypertrophy.—In hypertrophy of the heart, the actual size of "S" and its size relative to "R" assume great importance. Thus, when the mass of the right ventricle has become relatively larger than that of the left, "S" is extremely prominent in lead I, whereas the "R" of lead III becomes larger than that of lead II (normally the largest). Conversely, when the mass of the left ventricle has become relatively larger than the right, as in some cases of aortic, arterial or cardio-renal disease, "S" is extremely prominent in lead III, whereas the "R" of lead I has become larger than the "R" of lead II. These phenomena, the meaning of which has been put on a logical basis by the investigator previously referred to, have been confirmed by experimental and post mortem evidence, and are considered by Lewis as the most reliable mean

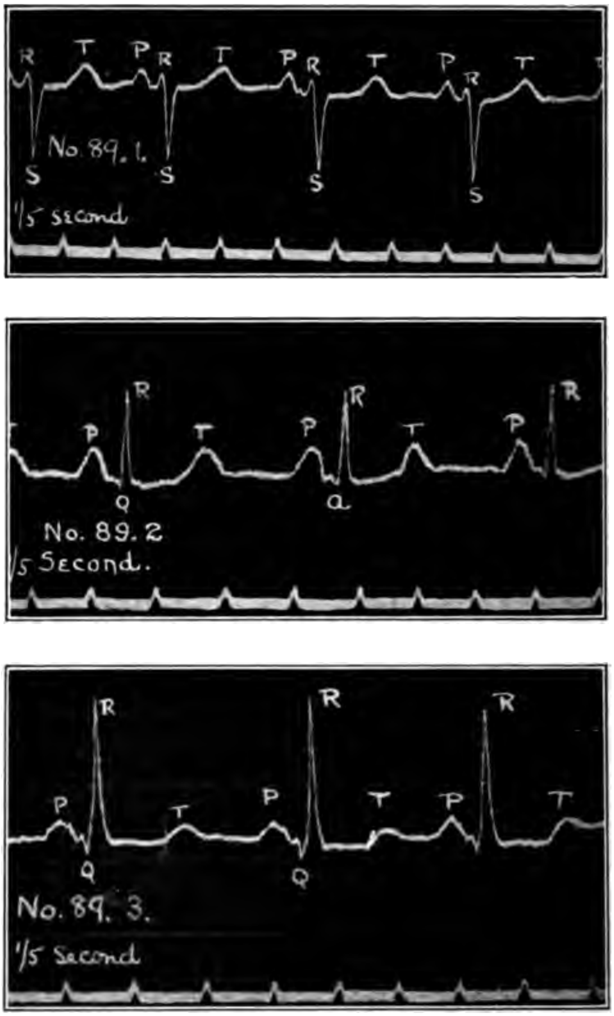

FIG. 146.—Preponderance of right ventricle. Curves from the three leads in a case o mitral stenosis, associated with auricular and right ventricular hypertrophy. In the ventricular complexes R_1 is almost absent and S_1 is very large, whereas R_3 is considerably large than R_2. Note the unusually large *P* of auricular hypertrophy. The black backgroun and white string of this illustration are due to the fact that the record is taken directly o sensitive paper. In this, as in a few others of the illustrations, some lines have been reinforced for purposes of reproduction. The magnification of the string shadow is less than i Fig. 145.

of estimating the relative size of the two ventricles. Clinical evidence that has been brought forward now and then to contradict this view has not been sustained, but it is certain at least that the form of the electrocardiogram is slightly influenced by the position of the heart in the body. (Compare the changes in form of the ventricular complex in deep respiration and after removal of large pleural effusions.) Electrocardiograms repeated on such cases over a period of several years will give valuable information as to the progress or stationary condition of the muscle changes in one or other ventricle.

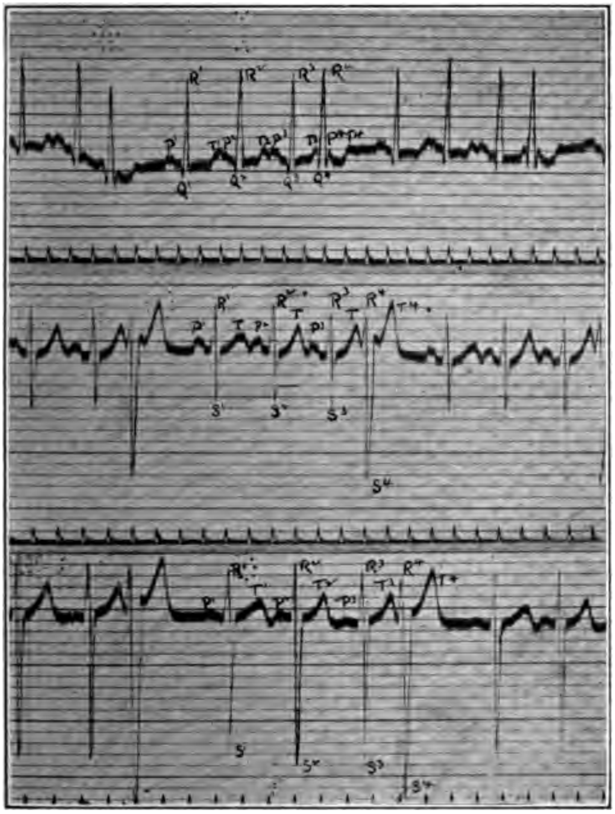

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FIG. 147.—Preponderance of the left ventricle. Note that R_1 is here higher than R_2, and that S_3 is the deepest of all. This case also shows interesting abnormal complexes, recurring in groups of four. It will be noticed that although the first three complexes of each group are rhythmical, yet the corresponding *P* and *T* waves of each group show constantly recurring small differences. The fourth complex is always premature, most probably a nodal extrasystole, and the whole represents a recurring "dislocation of the pacemaker," the origin of each of the 4 stimuli being different.

Congenital Heart Lesions.—In most cases of congenital heart disease, the electrocardiogram will merely show the result that the lesion has produced in the muscle balance (*i.e.*, there is usually a right ventricular preponderance) with exaggerated amplitude of all deflections. In dextrocardia, however, there is so great a change in the position of the heart in relation to the 3 customary leads that the form of the electrocardiogram is greatly changed. In Lead I, all deflections are completely inverted, and if the heart lies horizontally, Lead II will also be inverted. Lead III is usually normal. (With such records, especial caution must be observed that the electrodes are properly applied. It will readily be seen that accidental transposition of the electrodes to the two arms or leg might produce records that would be confused with that of dextrocardia.)

THE CARDIAC ARRHYTHMIAS

As the different types of cardiac arrhythmias have been set forth in the previous chapter, it will be necessary here only to indicate how they are manifested in the electrocardiogram.

Sinus Arrhythmia.—In sinus arrhythmia, the auricular summit and the ventricular complexes are of the normal supraventricular form; in other words, the impulse arises at the normal site and traverses the normal channels. That it is given out from the sinus, however, at irregular intervals, can readily be determined by measuring the varying intervals between the "P" summits.

Extrasystole.—Extrasystoles or premature contractions appear in the electrocardiogram in very striking guise. The ectopic origin of the stimulus is shown by marked changes in the shape of the deviations. Thus in auricular extrasystole, the normal "P" either becomes flattened out, diphasic or inverted. It is usually followed by a normal ventricular complex, and the pause before the next P wave is very rarely compensatory. Occasionally an auricular extrasystole may affect the succeeding ventricular complex, causing a so-called "aberrant" type. This may vary considerably or only slightly from the normal complex.

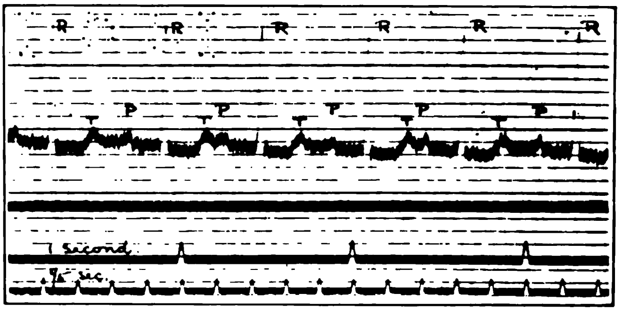

FIG. 148.—Sinus arrhythmia. Note that although all complexes are of normal shape, and the *P–R* interval is normal and constant, yet the *P* waves occur at irregular intervals, owing to arrhythmic stimulus production in the sinus.

Extrasystoles arising in the ventricle have been grouped into three main types, according to the site of origin. All are easily recognizable in the electrocardiogram and are characterized by the slowness and great amplitude of the deflections. Those arising near the apex of the left ventricle cause in lead I an extremely large deflection corresponding to "R" usually with an inverted "T"; in lead III, the deflection corresponding to "S" is unusually deep with an extra large "T." Extrasystoles arising near the basal portions of the right ventricle cause the reverse

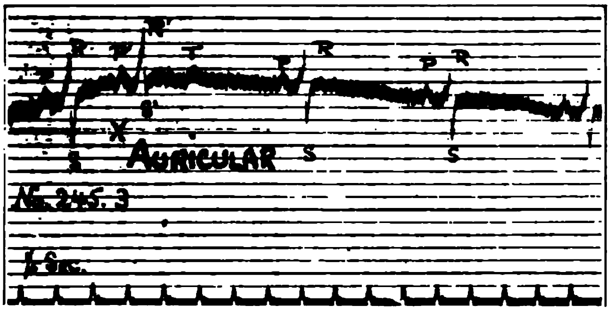

FIG. 149.—Auricular extrasystole. The normal rhythm is disturbed by the premature
earance of a *P* wave. In such cases it is frequently found to be of different outline than
normal or inverted. An auricular extrasystole is usually followed by a ventricular com-
of normal outline, but may give rise to an "aberrant" ventricular complex, as in the
sent instance. Note the larger *R* and relatively small *S* of the ventricular complex. The
t-extrasystolic pause is not compensatory, *i.e.* the distance from the first to the third
less then that from the third to the fifth (not lettered).

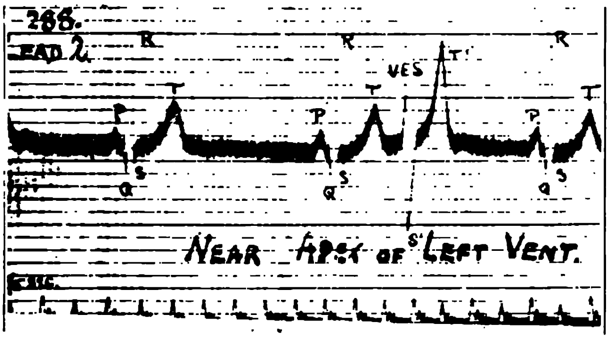

FIG. 150.—Ventricular extrasystole arising near apex of left ventricle. The deep down-
rd deflection and large *T* wave indicate that this extrasystole arose near the apex of the
ventricle. In lead III it would probably have had much the same form, and in lead I,
embled those of Fig. 154; although in some cases the form remains the same in all leads.
te that there is no compensatory pause in this instance; such an extrasystole is said to be
terpolated."

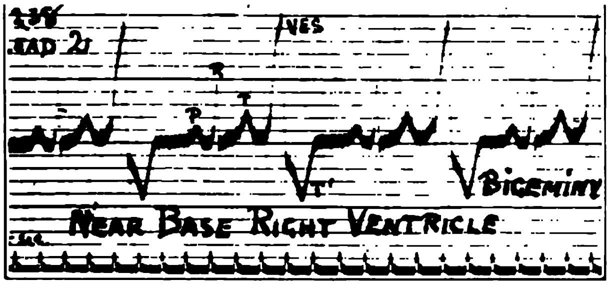

FIG. 151.—Ventricular extrasystole arising near base of right ventricle. Note the ex-
ne height of the initial deflection, with the exaggerated inverted *T*. The deflection time
he extrasystole is considerably longer than the normal. The extrasystolic complex in
, III was identical with this, whereas in lead I it resembled that of Fig. 153. The re-
ence of an extrasystole after each normal beat in this case causes a true bigeminy.

of this picture (*i.e.*, deep "S" and large "T" in lead I, and large "R" and inverted "T" in lead III). The site of origin of such extrasystoles has been proved by stimulation of these areas in animals with the production of similar electrocardiograms. Extrasystoles arising in the septum or intermediate positions give rise to a variety of anomalous complexes.

Extrasystoles arising in the junctional tissues (Tawara's Node and His' Bundle) are rare. In such a premature contraction, evidence of auricular activity occurs either very close to the ventricular complex (shortened "P-R" interval) or synchronously with the ventricular complex, which is of the normal or supraventricular shape. The "P" wave is usually diphasic or inverted on account of the changed direction of travel of the auricular impulse.

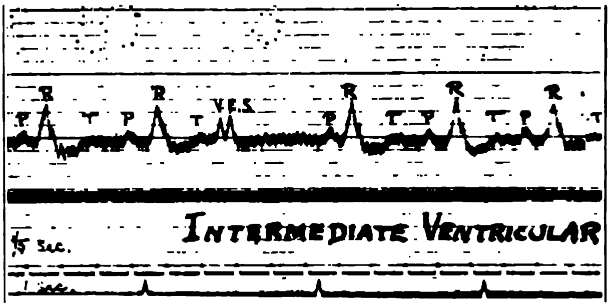

FIG. 152.—Ventricular extrasystole (intermediate). Extrasystoles arising near the ventricular septum appear in various intermediate forms, such as seen in the third complex of this illustration. The form of the *second* half of this extrasystole is undoubtedly influenced by the superposed *P* wave. Note that the fundamental auricular rhythm is undisturbed. The post-extrasystolic pause would be quite compensatory, were it not for the fact that the next succeeding *P–R* interval is shortened.

The site of the impulse formation of the premature contraction usually remains the same for long periods. In some cases, however, the anomalous impulse may arise in several locations ("dislocation of the pacemaker"). (See Fig. 147, where there are recurring cycles of four beats each. The stimulus for each beat of the four arises in a different spot, the last of the four being a nodal extrasystole.) If for any reason, the ventricular rate is very slow (as in partial block), the ventricle may occasionally "escape" and insert a beat either of supra- or intraventricular origin into the otherwise regular rhythm.

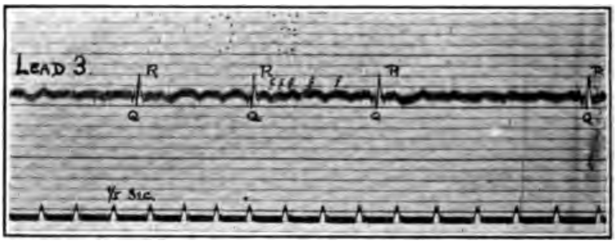

FIG. 153.—Auricular fibrillation. From a case of rheumatic mitral disease of long duration. Note the absence of *P* waves and the ventricular arrhythmia. The irregular waves of fibrillation occur over 300 to the minute, and obscure the *T* waves of the ventricular complex.

Auricular Fibrillation.—Fibrillation of the auricle is indicated in the electrocardiogram by three phenomena; first, the absence of "P," the sign of coördinate auricular contraction; second, the presence of low, rapid waves of fibrillation; and third, the irregular response of the ventricle. The fibrillation waves are best detected in lead III, or by a special lead from sternum to vertebræ. They may be barely discernible, fine or coarse. The ventricular complex is of supraventricular type, but may show such changes as those found in ventricular hypertrophy, deficient conductivity and so forth. While this condition is usually permanent, undoubted cases of paroxysmal or temporary fibrillation have been observed.

Heart Block. *Auriculo-ventricular.*—The various stages of auriculo-ventricular heart block are easily recognizable in the electrocardiogram. In the earliest stage (delayed conductivity), the time interval between "P" and "R" is found to exceed the normal limit of 0.2 seconds. In the next stage, "dropped beat," the "P-R" interval gradually gets longer until after several beats, a second "P" follows before the ventricle has responded to the first auricular impuls. On account of the varying "P-R" interval, accurate measurement shows a ventricular arrhythmia. Dropped beats may occur, however, without this gradual increase of the "P-R" interval. In partial block, every second, third or fourth "P" (2 to 1, 3 to 1, or 4 to 1 block) is followed by the ventricular response. In 3

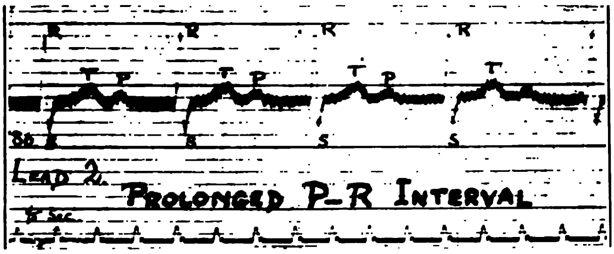

FIG. 154.—Auriculo-ventricular heart block. Prolonged *P–R* interval. From a case of paroxysmal tachycardia taken between paroxysms, the *P–R* interval is prolonged to 0.35 second. At other times, the *P–R* interval in this case was normal.

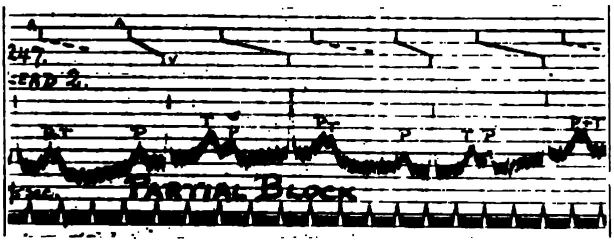

FIG. 155.—Auriculo-ventricular heart block. "Dropped beat." From a case of mitral stenosis, taken during a recurrence of acute articular rheumatism. The temporary occurrence of partial heart block in this case indicates an acute rheumatic myocarditis of the junctional tissues. With subsidence of rheumatic symptoms, the rhythm returned to normal, the *P–R* interval remaining prolonged for several days after the dropped beats had disappeared. Note here the regular recurrence of *P*, the gradual increase of the *P–R* interval, until *R* fails to respond, and the resulting ventricular arrhythmia. In spite of the occasional superposition of *P* and *T*, each wave can usually be identified by measurement, complete or partial.

to 2 rhythm, only one "P" in three is not followed by ventricular contraction. The grades of block may change from moment to moment, even while a record is being made. In complete block, "P" recurs at regular intervals, but bears no relation to the much slower, but also regular ventricular complex. The interpretation of these records is sometimes complicated by the coincidence of auricular and ventricular contraction. Careful analysis, however, of the summation of the electrical effects thus produced will show evidence of each of the individual peaks.

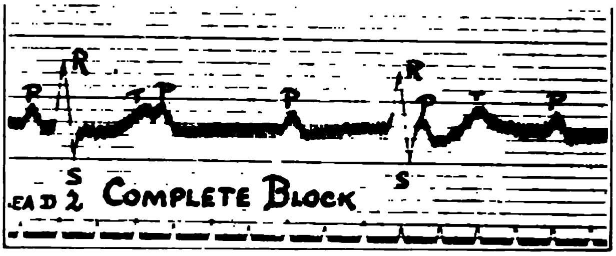

FIG. 156.—Auriculo-ventricular heart block. Complete block. Note that the *P* wave now occurs quite independently of the ventricular complexes, *i.e.*, there is no simple ratio between auricular and ventricular rate.

Intraventricular. A. Bundle Branch Block.—A fairly common type of heart block, that is only detectable by the electrocardiograph, is that occurring in one of the branches of His' Bundle. If one or other is deficient in conductivity, an anomalous complex results, which resembles that of right or left ventricular hypertrophy (*q.v.*), except that the QRS deflection time is considerably lengthened (more than 0.1 seconds), the initial deflections are of high amplitude, bizarre shape and notched.

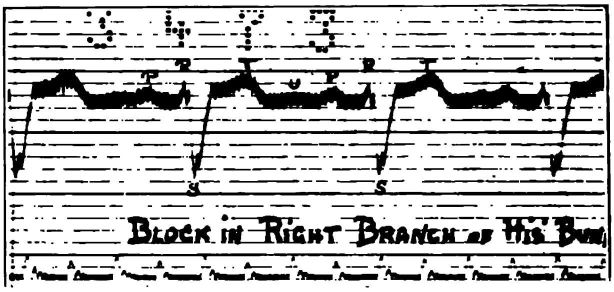

FIG. 157.—Intraventricular heart block. Right branch of His's bundle. From a case of decompensated myocarditis, which has been for two weeks on large doses of digitalis. Note the notching of *S* and the prolonged deflection time of the *R–S* group (more than 0.1 second). The *P–R* interval is prolonged to 0.24 second. As both these signs continued after the cessation of digitalis, they were presumably due to deficient conductivity caused by organic changes. Note also the small *U* wave that is occasionally seen in normal records.

There is usually a high T wave, in the opposite direction to the larger of the initial deflections. If the right branch is deficient and the impulse reaches the ventricle through the left branch, the picture resembles that of left ventricular hypertophy; and *vice versa* if the left branch is deficient. This condition may be a permanent result of organic disease or a temporary result of digitalis.

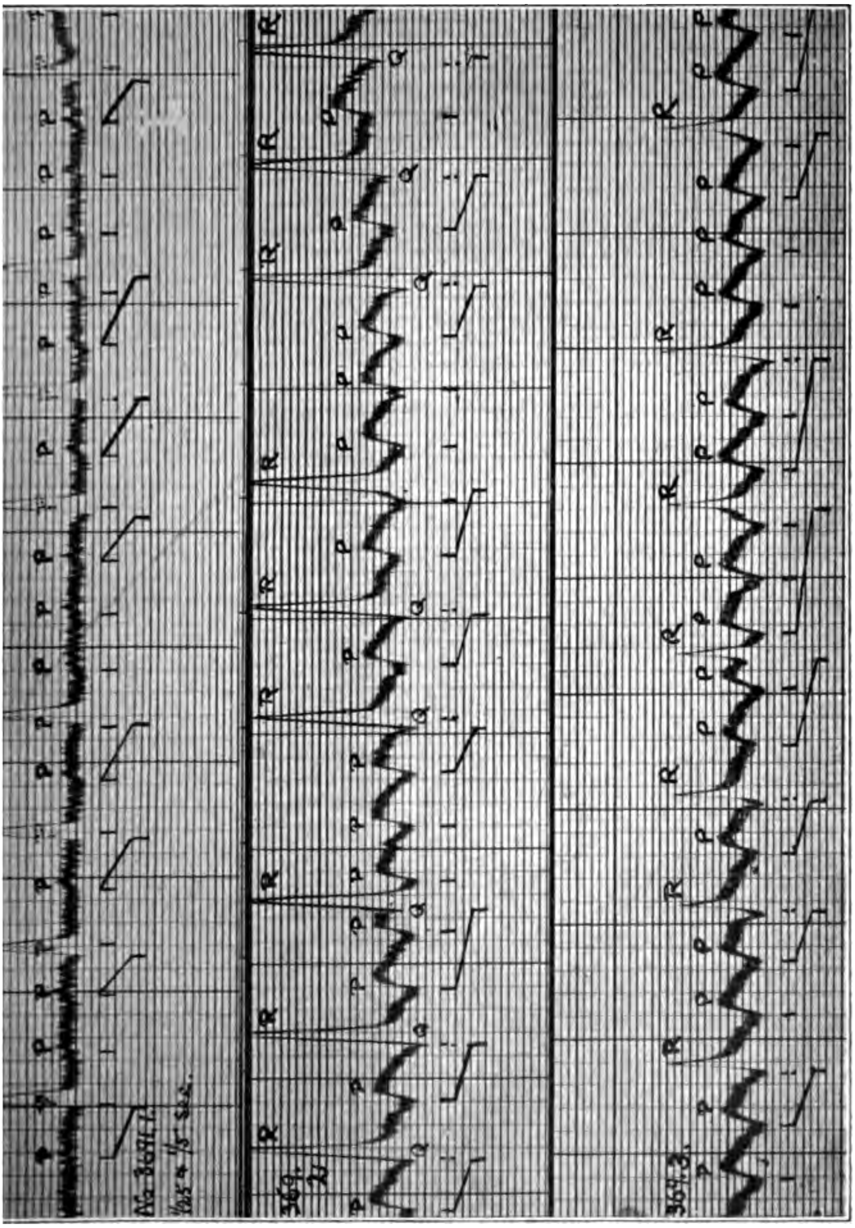

FIG. 158.—Auricular flutter. Curves from the three leads of a case of persistent auricu- flutter, under observation for over two years. The marked ventricular arrhythmia is to a constantly changing degree of heart block (2:1, 3:1, 4:1). For short periods a 3:1 k with regular ventricular rhythm was present. The auricular rate is over 300, the tricular about 120. Time intervals of $\frac{1}{5}$ and $\frac{1}{25}$ second are recorded by heavy and light tricular lines.

B. Arborization Block.—Oppenheimer and Rothschild have described abnormal electrocardiograms, which they consider to be due to interference with stimulus conduction beyond the two chief branches of the bundle of His, *i.e.*, in the arborization of the Purkinje fibers. Like bundle branch block, this condition shows the notching and prolonged duration of the initial deflections; but differs in the low amplitude of the initial deflections in all leads and in the absence of "typical diphasic curves" with huge T waves. This form of electrocardiogram is probably more frequent than that due to block of the main branch, and is important on account of its serious prognosis (Willius), and frequent association with coronary thrombosis.

Sino-auricular.—In the rare condition of sino-auricular block, occasional cycles are entirely absent (*i.e.*, there is no evidence of either auricular or ventricular activity). It is assumed in these cases that the sinus impulse has been blocked before reaching the auricle.

Auricular Flutter.—This relatively common form of arrhythmia was discovered by means of the electrocardiograph, and it is still impossible definitely to diagnose the condition without its aid. The ectopic origin in the auricle of the rapid stimulus production is usually shown by the inversion of "P" in one or other lead. If due allowance is made for the breaks caused by "R" and "T," a regular rapid succession of "P" summits can be traced through the record. As the ventricle cannot respond to all the impulses from the auricle, a state of partial block is present. If 2 or 3 to 1, the ventricular rhythm will be regular; if a 3 to 2 rhythm exists, the ventricular response will of course be irregular. This arrhythmia with the distortion of the succession of "P" summits by the superposition of occasional "T" summits, occasionally makes it difficult to diagnose true flutter from the coarse type of fibrillation.

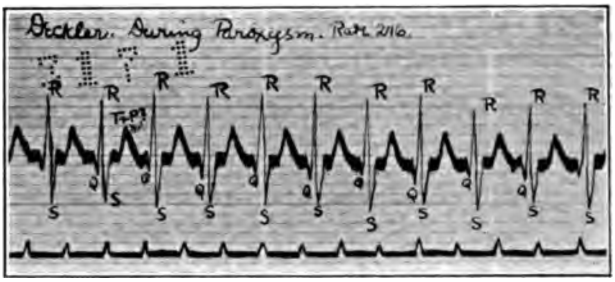

FIG. 159.—Alternation of initial deflections. Taken during an attack of paroxysmal tachycardia. Note the alternating height of *R* (this is further modified by changes due to respiration). Note also in each cycle that the bigger *R* is associated with a smaller *Q* and a bigger *S*. After the paroxysm alternation was no longer found. (N.B.—Alternation may also cause a similar difference in height of the T waves.)

Pulsus Alternans.—Many cases that show well-marked alternation in the arteriogram fail to show any abnormality in the electrocardiogram; evidence that the "R" peak is not a measure of contractility. Alternation of "R" may be present, however, in cases that may or may not show alternation in the arteriogram. This might be taken to show that alternation may result from deficiency in irritability or stimulus production as well as in contractility, but the question is still quite undecided. Considerable variation may occur in the height of successive "R" waves, without true alternation (*e.g.*, the changes in "R" due to forced respiration). If the respiratory factor can, however, be excluded, this never occurs in the normal heart, and though its presence is not necessarily of grave import, it must be taken as a sign of myocardial trouble. Alternation of T also occurs.

Paroxysmal Tachycardia.—Electrocardiographic records taken during a paroxysm of paroxysmal tachycardia, frequently show only two main deflections, "QRS" and a combination of "T" and the succeeding "P" (see Figs. 159 and 160). If these are separated, however, some abnormality in "P" usually betrays its ectopic origin (inverted, diphasic, or prolonged), if the paroxysm is of auricular origin. In some paroxysms, however, if the new site of stimulus production is near the sinus node, "P" almost or entirely retains its normal shape. The end of a paroxysm is shown by the *sudden* slowing of the cardiac rate, with the appearance of a normal "P" followed by a normal ventricular complex. Occasionally isolated auricular extrasystoles may disturb the normal rhythm for a short period after the end of the paroxysm, or the paroxysm may end in a short transition period of auricular or ventricular fibrillation.

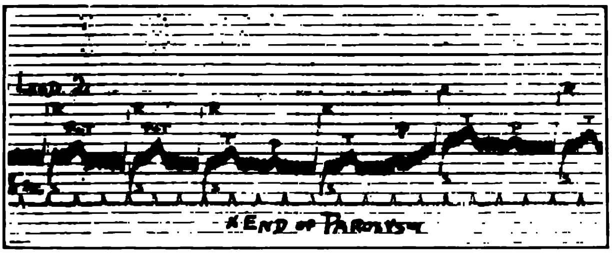

FIG. 160.—Paroxysmal tachycardia (auricular). Three beats of the paroxysm are reproduced to the left. The *P–R* interval is prolonged and *P* and *T* are superimposed. The rate is about 120; the rhythm suddenly drops to about 70, the *P–R* interval remaining prolonged. When the *P* waves of the paroxysm are visible, they are usually found to be of abnormal shape or inverted.

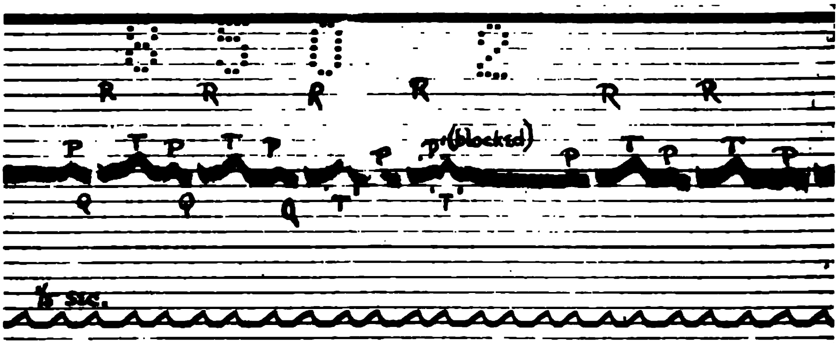

FIG. 161.—Blocked auricular extrasystole. The normal rhythm is interrupted by a long pause, and it will be noted that the *T* wave of the preceding systole is altered by the superposition of the auricular extrasystole (P^1). That this (and not sino-auricular block) is the correct interpretation, is confirmed by the fact that the alteration of the *T* wave is constant, and that after the administration of atropin, the auricular premature contraction is invariably followed by a ventricular contraction which partially obliterates the pause.

Paroxysms due to heterogenetic impulses arising in the ventricular or junctional tissues are rare. The former are characterized by the anomalous form of the ventricular complex; the latter by the very short or absent "P-R" interval.

Combined Arrhythmias.—A combination of two or more of these arrhythmias may occur in a single record. Thus in cases of auricular fibrillation the dominant complex of "supraventricular" origin, may occasionally be replaced by a beat of "intraventricular" origin, indistinguishable in form from a ventricular extrasystole or premature contraction. In the coupled beat of digitalis, the second beat of each pair is of this character. Just as the ventricle may "escape" if its irritability reaches too high a level, so the impulse from an auricular extrasystole may be blocked if it reaches the ventricle when its irritability is still lowered by the preceding beat (refractory period). An example of this is given in the accompanying figure, which duplicates the picture of sino-auricular block, except for the fact that the T wave preceding the pause is altered by the superposed auriclar extrasystole.

Effort Syndrome.—Inasmuch as the Effort Syndrome is not primarily a heart condition, but rather the general response of an exhausted nervous system, it is not surprising that the electrocardiograph furnishes only what evidence might be expected from consideration of the preceding paragraphs. Arrhythmia is uncommon and when present is nearly always either of the sinus or extrasystolic type, and is apt to disappear with the recovery of the patient. The signs of preponderance of one or other ventricle (usually the right) are occasionally found, and some cases of dilated hearts show the small deflections of "R" and "S," such as are often associated with myocardial weakness (see p. 180).

CONCLUSION

From the above considerations, it will readily be seen that much of the information given by the electrocardiograph has not yet been properly appraised. In the analysis of cardiac arrhythmias, to be sure, most moot points have already been settled; but as an indicator of the condition of the heart muscle—the most important item in the prognosis and treatment of cardiac disease—the limitations and possibilities of the electrocardiograph have not yet been realized. It furnishes valuable evidence about the relative size of the two ventricles, locates the site of origin of abnormal stimuli and accurately determines the various time relations of the cardiac cycle. Repeated records give information in several ways about the progress of the disease and the response of the heart to digitalis. Like all other clinical instruments of precision, however, it is at the best but one of several aids to the discriminating physician.

CHAPTER XVII

PALPATION

THE CARDIAC IMPULSE

This term is applied to the impulse of the heart against the chest wall. The study of this phenomenon may be carried on by simple inspection, by palpation or by graphic records.

The occurrence of *the cardiac impulse marks the time of:* (1) ventricular contraction; (2) the beginning of the first sound; (3) arterial filling. It is felt as a slight movement under the chest wall, somewhat gliding and slightly lifting in character, of definite rhythm but uncertain beginning, whose intensity depends on (*a*) the rapidity and force of the ventricular contraction, but even more largely on (*b*) the shape of the chest—the extent to which the lungs overlap the heart; and finally upon (*c*) the amount and character of overlying soft tissue. Thus in deep-chested, or corpulent individuals and in recumbency, the impulse may be normally imperceptible; while in long-chested persons, especially in emaciated subjects, in children, during forced expiration, under excitement, or exertion, it is disproportionately strong. The point of perception is found lower by palpation than by inspection. The duration of the impulse is normally about one-third of a second.

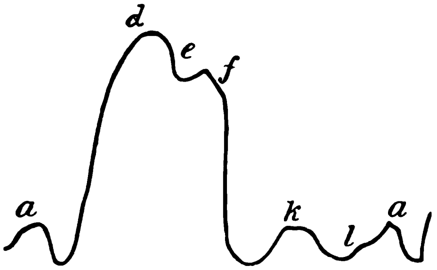

Fig. 162.—The normal cardiogram.

During ventricular systole the heart (1) becomes smaller, especially n its transverse diameter; (2) twists about its longitudinal axis from left to right and forward (Hirschfelder).

It has been graphically shown that what we see as the "apex beat" consists of variable and often complex elevations and depressions of the precordium. The movements may be grouped as:

1. A normal type: a considerable protrusion of the precordium, occurring with and during ventricular systole. Graphic tracings in such a case show three waves: (*a*) auricular systole; (*b*) ventricular systole; (*c*) ebound due to ventricular diastole (see Fig. 129).
2. Elevation of the whole precordium due to pivotal action of the

heart against the vertebral column. Seen chiefly in hypertrophied hearts, especially in flat-chested people.

3. Systolic retraction.

During systole, the right side of the right ventricle tends to recede from the chest wall. When this chamber is hypertrophied or acting forcibly, sufficient negative intrathoracic pressure is produced to cause a sinking in of more or less of the precordium. This normal retraction is sometimes mistakenly attributed to pericardial adhesions. When both elevation and retraction are coincidentally present over different areas, we get an impression of a wavy or peristaltic impulse. Retraction, elsewhere, may be caused by pericardial adhesions and when noted posteriorly

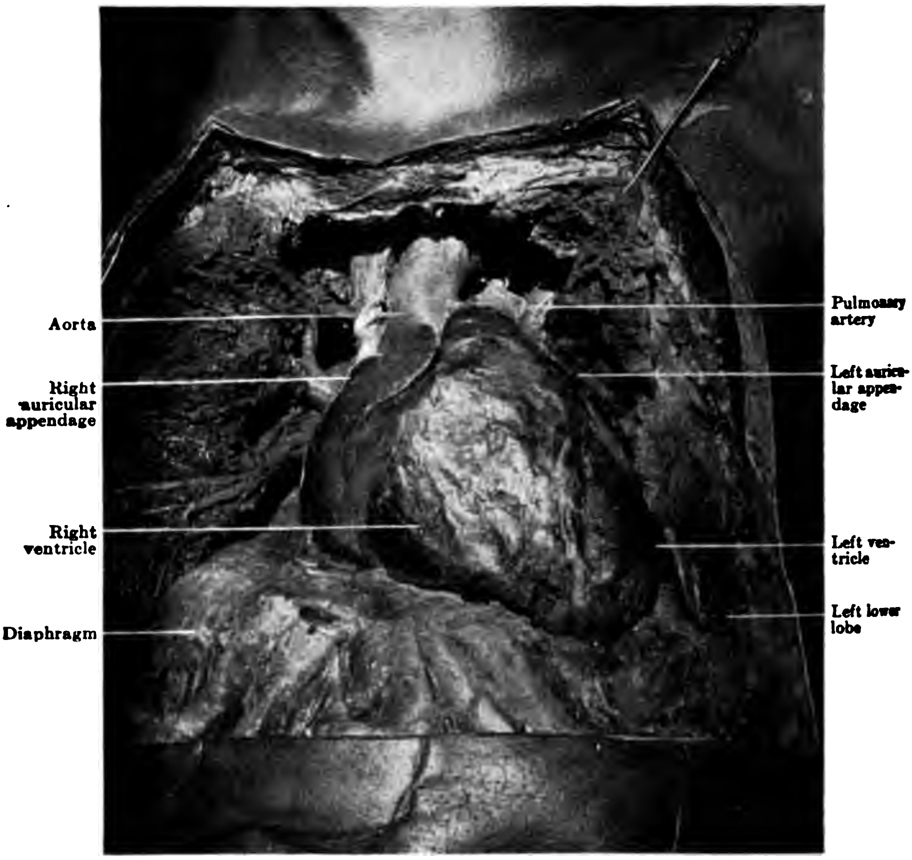

FIG. 163.—The cardiac impulse ("apex beat") does not correspond to the anatomic apex of the heart but is due to a sudden hardening and increase in tension of the anterior surface of the right ventricle, about 1 inch (2.5 cm.) to the right of the anatomic apex.

Practically the whole anterior surface of the heart, is the right heart. The left ventricle normally forms only the extreme left border of the anterior surface and comes in contact with the chest wall mainly when this chamber is enlarged, as is the case in the accompanying photograph, in which the whole heart, but especially the left ventricle, is greatly hypertrophied.

at the level of the eleventh and twelfth ribs is known as *Broadbent's sign* of adhesive pericarditis.

The examination of the cardiac impulse is of *diagnostic importance* since it enables us to estimate the degree of enlargement or displacement of the heart, especially to the left and downward, and often to determine whether enlargement is due to hypertrophy or dilatation. It is important, therefore, to know not so much the point of maximum intensity, but *the lowermost, outermost point of impulse.* It also enables us to accurately count the pulse in auricular fibrillation, etc., when the radial count is no longer a criterion of the pulse rate. It also renders possible the timing of heart sounds, murmurs, thrills, etc.

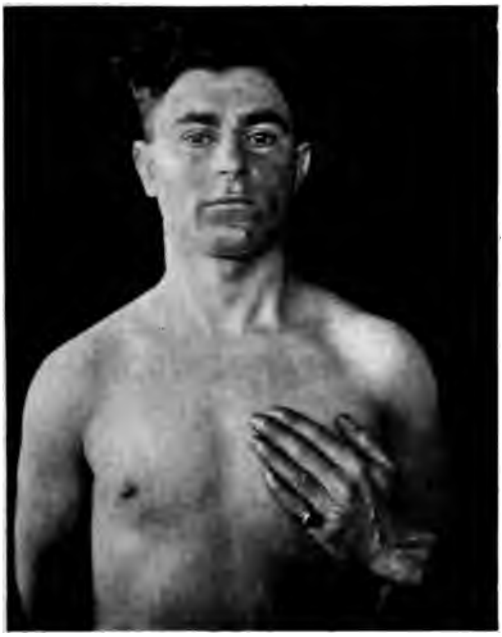

FIG. 164.—Palpation of the precordium. The hand should rest lightly upon the chest wall in order to determine the position and character of the cardiac impulse, or the existence of thrills.

Palpation of the cardiac impulse is generally more satisfactory than mere inspection. By placing a finger tip in an intercostal space, or by laying the whole palmar surface of the fingers lightly upon the precordium we not only confirm the data obtained by inspection but we may determine other important phenomena. In left ventricular *hypertrophy* a forcible, deliberate, prolonged heaving impulse is felt, while that of *dilatation* often has an abrupt, slapping quality. A heaving impulse, however, may be noted even in dilatation when compensation is far from good. The force of the *impulse is decreased* in obesity, emphysema, edema of the precordium, thickening of the lung or pleura, feeble heart action, cardiac dilatation, pericardial or pleural effusion.

Pulsation of the aorta may sometimes be noted in the second left intercostal space, but when marked should always suggest the possibility of aneurism (Fig. 432). The pulsation most commonly encountered at the base of the heart is that of the pulmonary artery. *Epigastric pulsation* generally indicates active right ventricular contraction or a low diaphragm. It is, therefore, most marked when this chamber is hypertrophied, or when general visceroptosis is marked. Pulsation in this region may result from abdominal aneurism, tumor, a relaxed aorta, or from a pulsating liver (tricuspid insufficiency). A *diastolic "shock"* over the base of the heart indicates a sudden, forcible closure of the semilunar valves. It may be felt when congestion of the lungs is associated with a vigorous right heart, and in aortic aneurism.

In referring to the *apex impulse* it is to be definitely understood we mean the *outermost, lowermost point at which it is either visible or palpable*, and not the point of maximum intensity. It is generally felt as a diffuse throb ¾ inch inside the left border of percussion dulness; and almost always one interspace higher than the silhouette obtained by the orthodiagraph: *firstly*, because it is felt and seen in systole, while the X-ray shows the heart in diastole (Dietlen); and *secondly*, because what is generally felt as the apex beat is *not due to a slapping of the anatomic apex against the thoracic parietes, but to a sudden marked increase in tension of the lower, anterior surface of the right ventricle.*

The left ventricle is a direct factor in its production only in case of hypertrophy of that chamber, or of cardiac displacement. "The anatomic apex, formed by the tip of the left ventricle, comes into direct contact with the thoracic wall only when the apex is situated far enough to the left to strike the lateral wall of the chest" (Dayton).[1]

Normal Position of the Cardiac Impulse.—The cardiac impulse is normally observed (in adults) in the fifth interspace from 9 to 10.5 cm. from the mid-sternal line as an ill-defined and sometimes distinctly wavy elevation and retraction of the intercostal space. From what has been stated it is evident that the position and character of the cardiac impulse will vary greatly in different individuals. Such variations may result from a *change of posture:* the impulse is generally less marked in recumbency, and can often only be felt when the individual is erect and leans forward; sometimes in normal cases not even then. The assumption of the left lateral decubitus causes it to move 1½ to 2 inches to the left; lying on the right side causes less displacement. The failure of the impulse to move with a change of posture may be due to pericardial adhesions

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[1] Numerous physiologists teach that the apex impulse is due to the thrust of the left apex against the chest wall, the heart being rotated on its axis in the process of contraction, so as to bring the left ventricle into an anterior position. In defence of the statement made by us the following facts may be mentioned: (1) The left ventricle in the normal heart is a posterior structure, only a narrow margin of which is visible from in front. (2) The cardiac dulness always extends at least ½ inch to the left of the outermost portion of the apex impulse, showing that the impulse is not produced by the anatomic apex of the heart. (3) The fact that the exposed heart can be seen to twist upon its axis during systole is not proof that the normally surrounded heart performs the same movement. "The heart is not tilted forward to the right during systole; this occurs only when the heart has been displaced on opening the chest." (4) "As the heart expands during diastole it becomes at the same time so flaccid as to flatten by its own weight; it therefore becomes distorted by the ever-changing walls of the cavity in which it rests (chest walls and lungs) and during systole its first act is to recover itself, 'asserting itself, against the tissues which surround it.' This causes the apex beat" (Haycroft, J. B.: "The Movements of the Heart within the Chest Cavity and the Cardiogram," *Jour. Physiol.*, 1891, 438).

'igs. 218, 380, 381). Excessive motion "*cor mobile*" (3 to 5 inches) oc-
rs in cardioptosis, and is associated with a low diaphragm, and often
th visceroptosis and arterial hypotension. The cardiac impulse is higher
children, and lower in old age, than in adult life. A feeble impulse, like
faint heart sound, by no means indicates a weak heart action. It points
ther to large lungs, a deep chest or thickness of the superficial tissues.

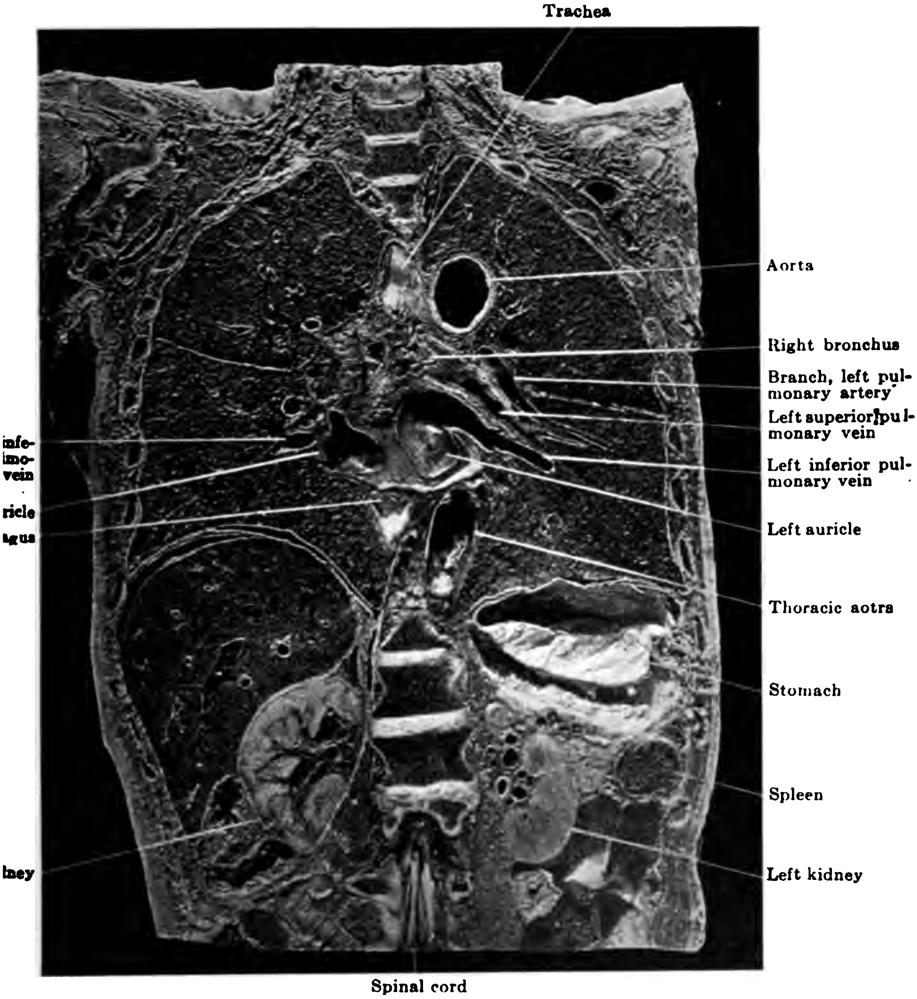

FIG. 165.—THE POSTERIOR PART OF THE MIDDLE MEDIASTINUM. Showing the anatomic
lations immediately behind the heart viewed from in front. Compare Figs. 91 and 92,
owing the rear view.

Displacement of the Cardiac Impulse.—The cardiac impulse may be
isplaced *downward* if the diaphragm occupies a low position (cardi-
ptosis, visceroptosis) and in left ventricular hypertrophy. It may be
und to the *right* of its normal position in pericardial effusions, left-

sided pleural effusions or pneumothoraces, or as a result of right-sided pulmonary adhesions or fibrosis, as well as in situs inversus viscerum (Figs. 215, 216, 217).

"The character and degree of cardiac displacement in cases of *pericardial effusion* depends mainly upon the degree of compensation—the degree of increased venous pressure—in other words upon the specific gravity of the heart. If venous pressure is considerably higher than intrapericardial pressure, the heart will contain a normal amount of blood and remain in its normal position; if venous pressure is low, the heart will contain but little blood and hence will be pushed upward and backward, the apex deviating to the right"[1] (see Figs. 377, 378, 379).

The impulse may be displaced to the *left* in hypertrophy or dilatation of the ventricles, especially the left; or by a right-sided pleural effusion or pneumothorax and as a result of left-sided pulmonary fibrosis.

Dislocation in an *upward* direction may be caused by a high diaphragmatic position—meteorism, ascites, pregnancy, massive abdominal tumors—or by fibrosis of the upper pulmonary lobes.

THRILLS

A thrill is the *tactile perception of vibrations produced by flowing liquid.* It generally consists of intermittent vibrations of the chest wall, not unlike the sensation imparted by a purring cat; but the vibrations of a thrill are much finer and more rapid. Experimentally thrills may be produced by constricting a rubber tube or a blood-vessel through which fluid is flowing. They are due to vibrations of tissues, the mechanism being similar to that which produces murmurs (see p. 227). They may disappear if blood flow is insufficiently rapid to throw the tissues into vibration—low pressure, weak heart. Like murmurs, they may be conducted by the blood stream or the vessel walls. The hand must be placed very lightly upon the chest wall, since firm pressure may abolish the vibrations, which are generally produced by the eddies in the blood stream, caused by localized constrictions or dilatations of the heart chambers or vessel walls, *e.g.*, mitral obstruction, aortic aneurism.

In studying a thrill *it is essential to determine:* (1) the exact time of the cardiac cycle at which it occurs. This is accomplished by noting the relation of the latter to the apex impulse, (2) the location, and (3) the extent of the thrill.

Normal Thrills.—These occur when heart action is vigorous and the chest wall is thin—children, emaciated subjects, retracted lungs. In these cases the first heart sound is often loud and somewhat rasping, and in children the pulmonic second sound, loud. These signs may lead to an erroneous diagnosis of mitral obstruction, especially since with a rapid heart action a normal systolic thrill may be interpreted as being presystolic, and inasmuch as a normal presystolic sound is sometimes audible (see p. 221).

Such cases may be differentiated from mitral obstruction by the fact that in the latter condition the heart is enlarged, the pulse volume small, the rate not infrequently irregular, while the pulmonic second sound is unduly accentuated. In addition to these signs the patient with a stenotic lesion is apt to suffer from dyspnea, cyanosis, or cough on severe exertion.

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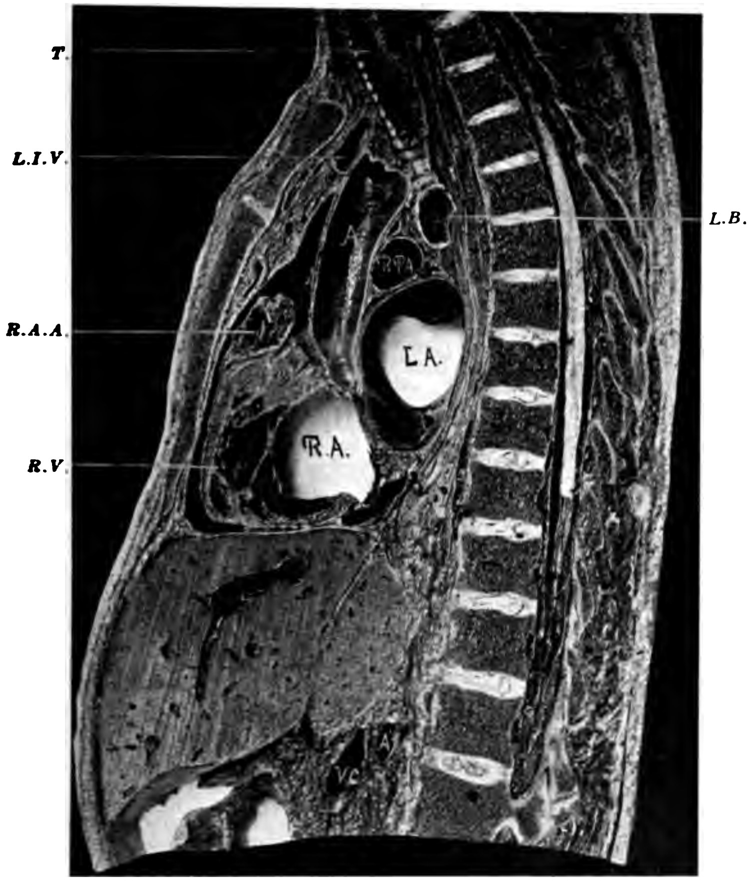
[1] NORRIS, G. W.: "Studies in Cardiac Pathology," 1911, 123.

'athologic Thrills.—Thrills of pathologic origin are encountered in ular heart disease and in aneurism. Thrills may also be felt in large arteries and veins, as well as over the thyroid gland in exoph-



ig. 166.—SECTION THROUGH THE MIDDLE OF THE STERNUM AND VERTEBRAL COLUMN lt). Showing the anatomic relations of various cardiac structures. At this point the t occupies practically the entire mediastinal space. The esophagus lies closely behind left auricle. Venous tracing depicting the functional activity may be made by introng a rubber capsule into the esophagus. *T.*, trachea; *L.I.V.*, left innominate vein; *A.*, right auricular appendage; *R.V.*, right ventricle; *A.*, aorta; *R.P.A.*, right pulmo-artery; *L.B.*, left bronchus; *L.A.*, left auricle; *R.A.*, right auricle; *A*, abdominal aorta; ., inferior vena cava.

mic goitre. The most intense *systolic* thrills are generally met with ortic obstruction, and pulmonary stenosis. Very marked *diastolic* ls are frequently encountered in aortic insufficiency and mitral ruction.

CHAPTER XVIII

PERCUSSION OF THE HEART

The heart is outlined by percussion *in order to determine:* (1) the size of the organ as a whole or the relative size of its different chambers; (2) its position; (3) the size of the great vessels at its base.

Method.—(1) Begin percussing below the left clavicle and continue downward until the upper border of cardiac dulness is reached. This point is indicated by a diminution of pulmonary resonance and will generally correspond to the upper border of the third rib. (2) Percuss from the left mid-axillary line toward the heart and note the point at which, over different ribs or interspaces the percussion sound becomes

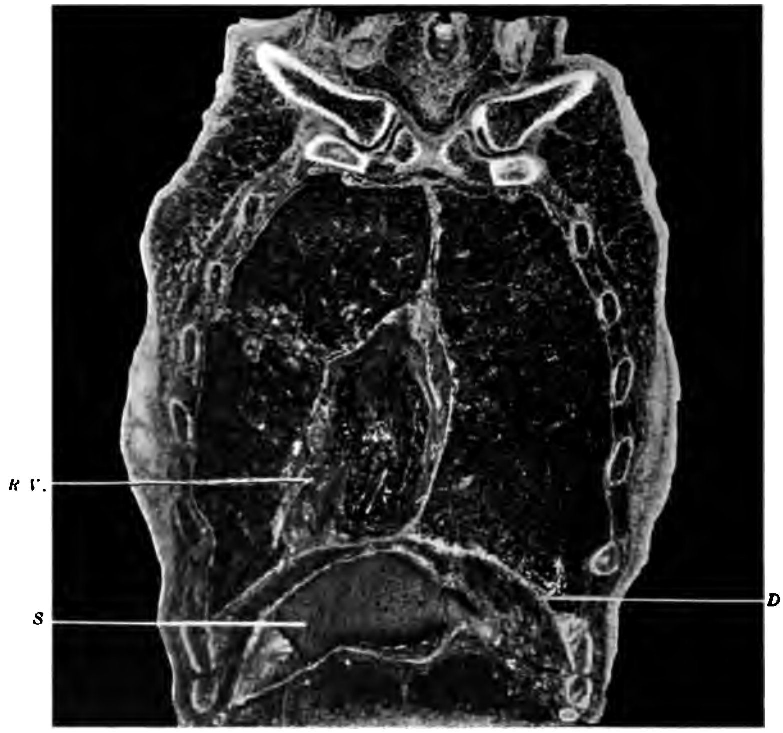
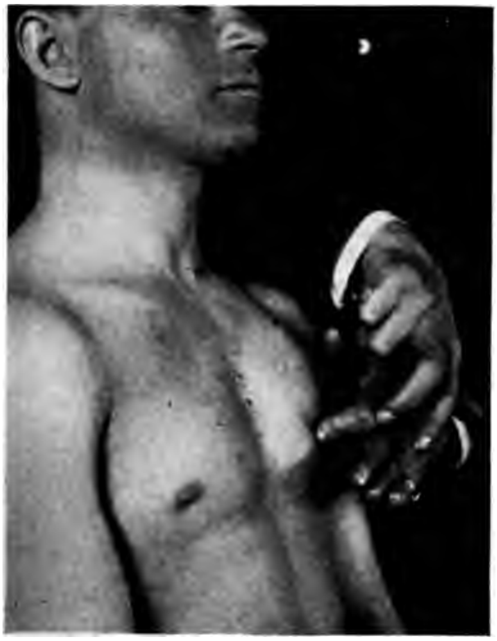
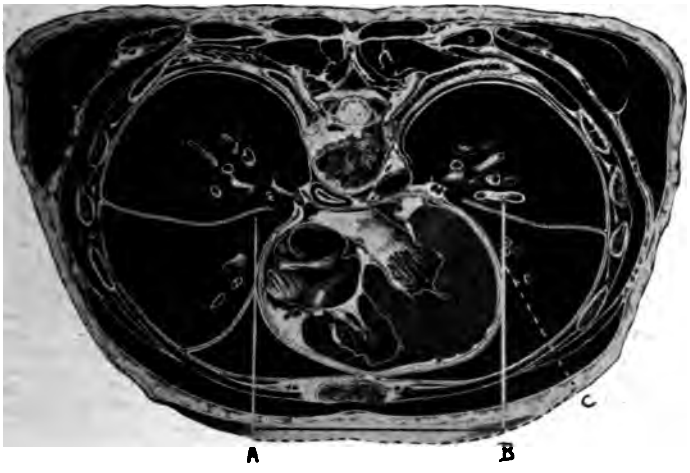


FIG. 167.—The anterior chest wall viewed from behind. This, the first of a series o sections, shows the anterior wall of the heart viewed from within, surrounded by the lungs. *R.V.*, anterior wall of right ventricle; *S*, stomach; *D*, diaphragm.



IG. 168.—**Orthopercussion.** The tendency is for the examiner to underestimate the
, **and overestimate the left,** border of the heart. This error may be minimized by
; **ortho-percussion with** a very light stroke.



IG. 169.—Horizontal section through the chest. Showing the unavoidable margin of
in outlining the left cardiac border by percussion. The solid line indicates the ver-
penetration of the orthodiagraph. The dotted line illustrates the oblique penetration
rcussion vibrations. *A–B*, orthodiagram shadow. *A–C*, percussion dulness. Light
cal percussion (orthopercussion) minimizes lateral radiation and gives the most ac-
te results attainable by percussion. The tendency is generally to overestimate the
and to underestimate the right cardiac border. The margin of error is increased in
of left ventricular enlargement and in patients with deep thoraces. The drawing
er shows the deep situation of the mitral, and the relatively superficial position of
ricuspid valves. (*After Braune.*)

slightly dull. (3) Percuss from the right mid-clavicular line and outline the right border in a similar manner.

The points at which the clear pulmonary resonance becomes impaired should be marked by means of a series of dots with a skin pencil or ink. Connecting lines between these dots should not be drawn until after the examination is complete, lest expectancy warp one's judgment. The *right border* requires the heavier percussion; the heart at this point recedes from the chest wall and its location is often determined with difficulty. Dulness normally extends slightly beyond the right sternal

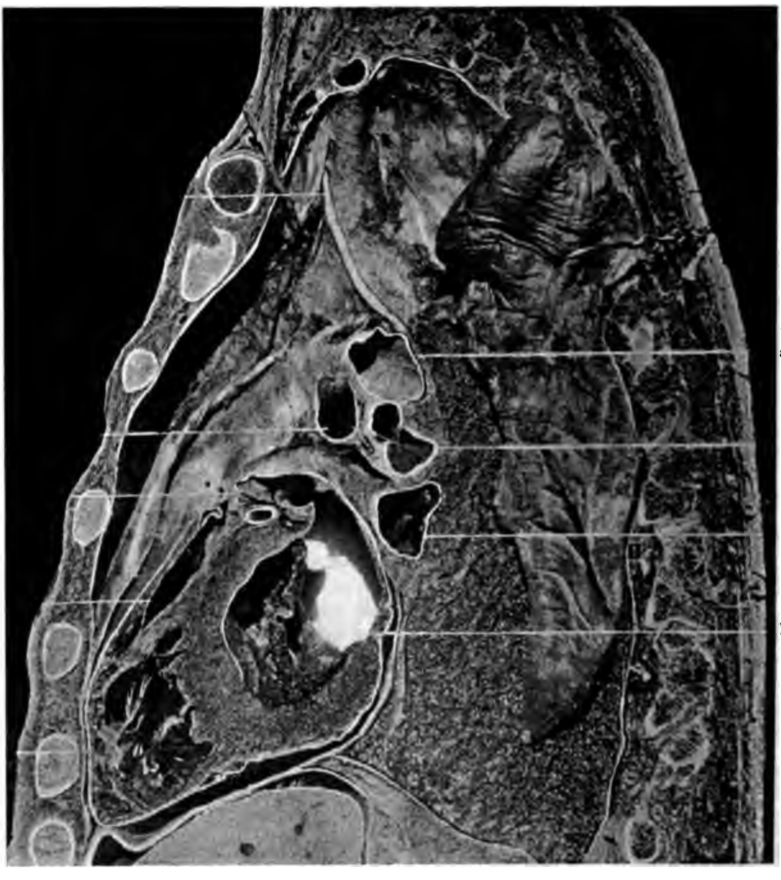


FIG. 170.—Vertical antero-posterior section through the heart showing this organ from a lateral aspect. In following the curve of the ribs during percussion from the sternum to the axilla, if forcible percussion is used the cardiac dulness obtained will be equivalent not only to the lateral boundary of the organ (which we wish to determine) but also to the depth or antero-posterior diameter (which vitiates the results). The only part of the left auricle which approaches the anterior chest wall is its appendage, which juts around the pulmonary artery and if greatly enlarged may cause an increase in dulness upward and to the left. (Compare Fig. 163.)

border, but the position of the right-sided dulness varies greatly in health. Very light percussion should be used for the *left border* and especial care should be exercised to deliver the percussion blow in a vertical direction (see Fig. 80).

Unless the percussion is performed with great care, the left border of the heart will be over-, the right border, under-estimated. In the majority of cases percussion of the cardiac outline by a skilful examiner yields results which are sufficiently accurate for most clinical purposes. Such accuracy consists, however, of variations in centimeters, not millimeters. In some instances, however, other methods of examination such as orthodiagraphy and teleoroentgenography must be employed and in all instances percussion should be controlled by palpation, it being borne in

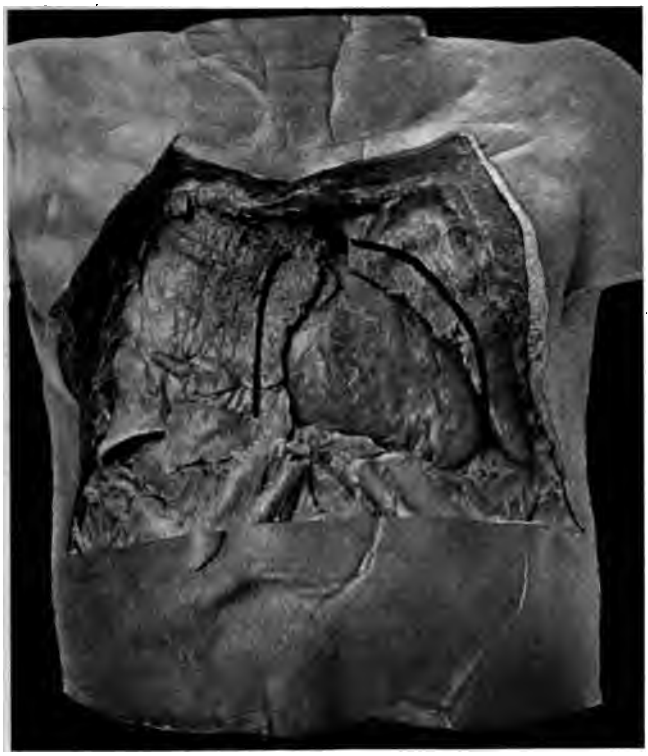


FIG. 171.—The deep cardiac dulness corresponds to the actual size of the heart. In percussing, we generally overestimate the extension of the left border since the slope of the ribs causes the percussion stroke to fall upon the side of the heart instead of solely upon its anterior surface. The extension of the right border, on the other hand, is generally underestimated since it slopes sharply away from the chest wall and is overlain by a thick section of lung tissue. The superficial (absolute) cardiac dulness represents the portion of the heart which is uncovered by lung tissue. This photograph depicts an unusually large superficial dulness.

mind that the left border usually extends 1.5 cm. beyond the lowermost and outermost point at which the heart impulse can be felt.

The portion of the heart which is uncovered by lung yields a flat percussion note. This area is known as the *superficial cardiac dulness*. It is of minor importance since it tells us more concerning the state of the lungs than that of the heart. The *deep cardiac dulness*, on the other hand, corresponds to the size of the heart. It may be determined by light or moderately heavy percussion, by noting the points at which clear pulmonary resonance ceases and slight dulness begins. A distinct increase of resistance will be noted at similar points. *Light percussion* is generally *preferable* since there is less radiation of the vibrations and a keener perception of them both by hearing and touch.[1] The *superficial area of dulness* is absent in animals and in some people with long thoraces and freely movable hearts, also in barrel-shaped chests with large lungs—emphysema. It is enlarged in flat-chested individuals, in tuberculosis—retracted lungs—in some rachitic deformities and in pericardial effusions. The *cardio-hepatic angle* is normally acute, with its concavity toward the right. It becomes obtuse, with a convexity toward the right in pericardial effusions and in right ventricular dilatation. In the latter condition an interrupted line—staircase form—has been described.

The *lower cardiac border* cannot be outlined by percussion because the heart and the liver overlap, and being structures of relatively equal densities both yield a similar note. Furthermore, the proximity of Traube's space (Fig. 89) adds a tympanitic element to the sound and as we have already learned, vibrations cannot be localized in tympany producing organs. Orthodiagraphic as well as post-mortem studies have shown that the heart can be very accurately outlined by careful, skilful percussion; the margin of error should in the average case not exceed 1 cm.

THE SIGNIFICANCE OF CARDIAC DULNESS

The different diameters of the heart bear a definite relation to the age, sex and height of the individual. Disproportionate enlargement of certain diameters is quite characteristic of certain cardiac lesions. A satisfactory method of recording the cardiac dimensions is shown in Fig. 173. *The area of heart dulness is normally from* 10 *to* 30 *per cent. larger in the horizontal than in the erect posture.* This is due to flattening of the chest and decreased backward convexity of the dorsal spine in the former position. In the erect posture the heart tends to fall away from the chest wall. Furthermore, the heart contains more blood in recumbency, owing to a higher venous pressure; its systolic output is larger. The left border of the heart is in adults; usually situated from 9 to 10.5 cm. to the left of the mid-sternal line.

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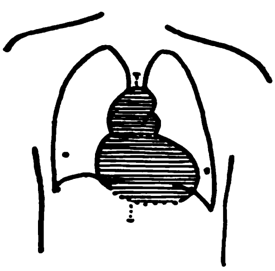
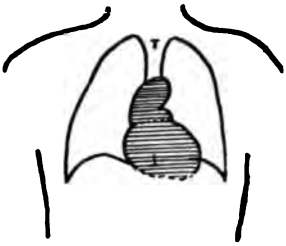
[1] Moritz found that with light percussion for the right and threshold percussion for the left, cardiac border, the former corresponded with the orthodiagraphic findings in 86, the latter in 70, per cent. of his cases.

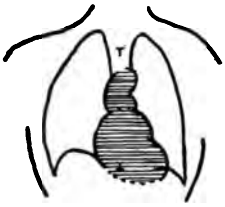
FIG. 172.—HEART DULNESS AS MODIFIED BY DISEASE. These figures illustrate the more or less characteristic shape and size of the heart as a result of different cardiac lesions. They are drawn after radiograms and correspond to the areas of dulness which would be obtained by skilful percussion. (*After F. M. Groedel.*)



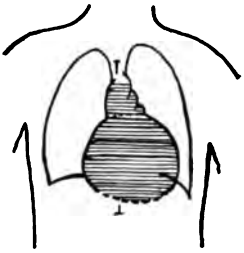
Aortic insufficiency.



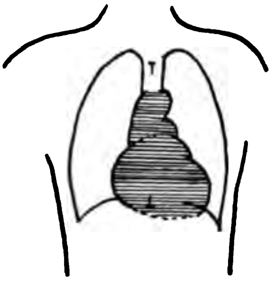
Aortic obstruction.



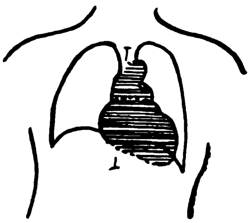
Mitral obstruction.



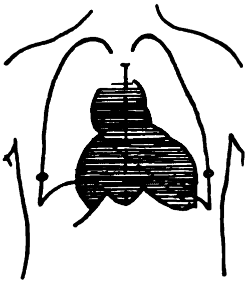
Mitral insufficiency.



Mitral obstruction and insufficiency.



Aortic obstruction and mitral insufficiency.



Chronic myocarditis.

FIG. 172.

Enlargement toward the left indicates left, sometimes right, ventricular enlargement. An increase of dulness *to the right* of the sternum may be due to right auricular dilatation or to pericardial effusion. An enlarged area of dulness *at the base* is often the result of aortic dilation or aneurism. The left auricle being a posterior chamber cannot be outlined by percussion. Marked enlargement of the left upper cardiac dulness may result from dilatation of the left auricular appendix which lies just to the left of the pulmonary artery (see Fig. 163). X-ray studies have shown that simple mitral insufficiency never causes dilatation of the left auricular appendix, but in mitral stenosis with insufficiency and in mitral insufficiency during broken compensation (tricuspid insufficiency) both dilatation and pulsation are demonstrable.[1]

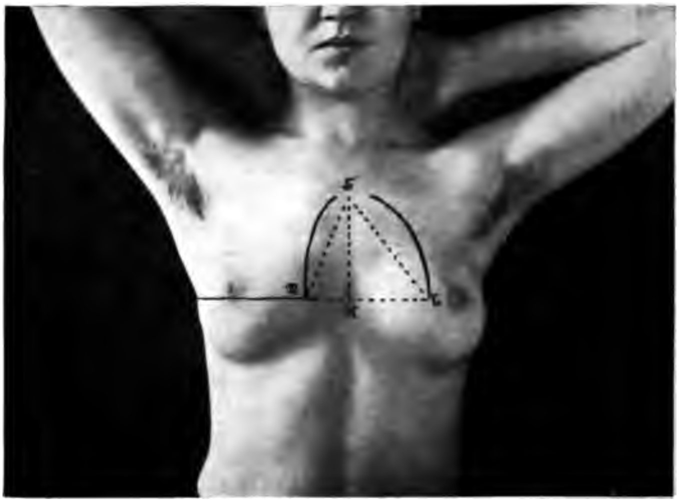


FIG. 173.—Method for recording cardiac dulness.

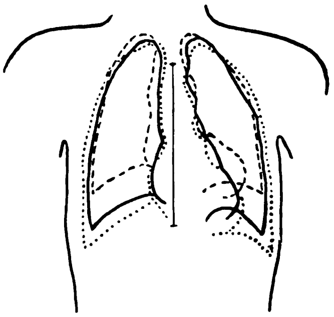


FIG. 174.—THE INFLUENCE OF RESPIRATION UPON THE POSITION OF THE HEART. (*Groedel.*) Heavy line: normal breathing. Interrupted line: deep expiration. Dotted line: deep inspiration.

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[1] PESCI, G.: "L'augmento di volume della brecchietta sinistra del cuore nel quadro radiologico." *Radiologia med.*, I, 1914, 106.

ncreased dulness at the base is generally due to dilatation or aneur-
)f the aorta (Figs. 409, 412); it may in rare instances be due to a lous ductus arteriosus. This lesion produces a quadrilateral area of ess to the left in the second (and first) interspace. Accurate outlining ercussion of the normal aorta and pulmonary artery is difficult and ıeous results are frequent, owing to the overlying sternum.

1ethod of Recording the Size of the Heart.—After outlining the heart ercussion and drawing lines upon the skin to correspond with the of dulness, the following dimensions should be measured and recorded. line is drawn from the cardio-hepatic angle to the lowermost portion ıe left border. A second line is drawn from the upper border of

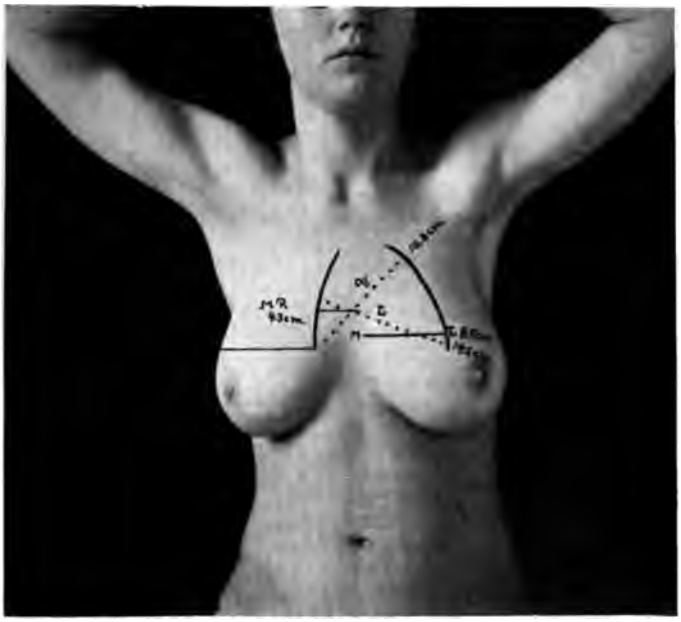

FIG. 175.—Moritz's conjugates.

liac dulness down the middle of the sternum to connect with the first . The extremes of these lines are connected and the following dimen-
s recorded in centimeters.

Height (S. H.); Left oblique (L. S.); Right oblique (R. S.); Right (R. H.); Left base (L. H.) (see Fig. 173).

These dimensions undergo characteristic variations in different valvu-
esions, owing to a disproportionate increase in the size of different nbers. Thus the *left base* is increased in left ventricular hypertrophy in mitral insufficiency. The *right base* is increased in tricuspid lesions dilatation of the right auricle. The height is increased in aortic se, etc. (Fig. 172).

Moritz has suggested a somewhat different method of recording the iac area which has found considerable favor, the dimensions thus ob-
ed being known as *Moritz's conjugates* (Fig. 175).

It has been shown by a large number of orthodiagraphic studies that the normal heart bears a fairly constant relation to the height of the individual. The normal averages are shown in the following table.

Height of individual		Men					Women				
Cm.	Feet and inches	*MR* Cm.	*ML* Cm.	*L* Cm.	*Q* Cm.	Cardiac area, *Q* Cm.	*MR* Cm.	*ML* Cm.	*L* Cm.	*Q* Cm.	Cardiac area, *Q* Cm.
145–154	4.7–5	3.5	7.9	12.5	9.7	95	3.5	8.1	12.7	9.4	93
155–164	5.1–5.5	4.1	8.7	13.8	9.9	109	3.5	8.4	13.2	9.7	101
165–174	5.5–5.9	4.2	8.8	14.1	10.3	116	3.8	8.5	13.4	9.9	105
175–187	5.9–6.2	4.4	9.1	14.8	10.7	127					

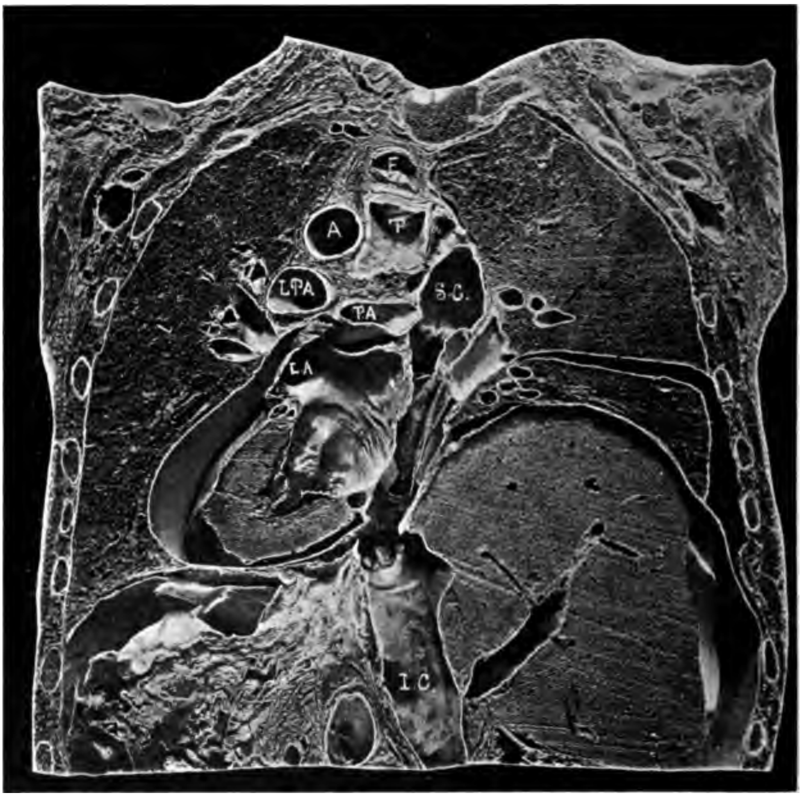

FIG. 176.—PERICARDIAL EFFUSION. *Viewed from behind*, causing enlargement of cardiac dulness to the left and compression of the left lower pulmonary lobe. The *left ventricle* is hypertrophied, the mitral valve sclerotic. *The liver* is enlarged and upward pressure from ascitic fluid has caused compression of the right lower lobe. *A*, aorta; *T*, trachea; *E*, esophagus; *P.A.*, right pulmonary artery; *L.P.A.*, left pulmonary artery; *S.C.*, superior vena cava; *L.A.*, left auricular appendage; *I.C.*, inferior vena cava. (Compare Fig. 216.)

The Orthodiagraph.—The ordinary X-ray plate is inaccurate for the purpose of determining the size of the heart. The reason for this is the fact that the heart lies from 7 to 10 cm. from the anterior chest wall.

It is, therefore, further from the source of light than are the anterior ribs and since the X-rays are divergent the heart shadow varies inconstantly and disproportionately in relation to the ribs.

The orthodiagraph overcomes this difficulty by allowing only perpendicular rays to fall upon a fluorescent screen and hence a parallel projection is obtained. The patient is placed between the source of light and the screen, and the outline cast by the shadow of the heart in its different dimensions is plotted upon the screen from which it can be accurately reproduced and measured.

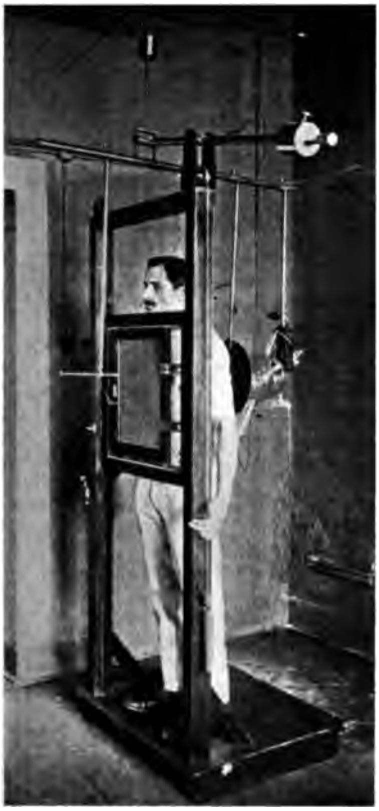

FIG. 177.—The orthodiagraph. (*Hoffman.*)

Almost equally accurate results may be obtained by *teleoroentgenography, i.e.,* photographing the heart by placing the plate over the precordium the tube being behind and at a distance of two meters from the patient, thus practically eliminating distortion.

CHAPTER XIX

AUSCULTATION

In ausculting the heart, the stethoscope is far preferable to the unaided ear since we are better able to localize sounds. The bell of the stethoscope is placed especially over certain areas of the chest wall at which the sounds produced at different valvular areas are best heard. These areas are named after the valves in question. It is to be remembered that these valvular areas do not correspond to the anatomic location of the valves. Anatomically the valves lie very close together (see Fig. 125).

The heart is auscultcd to determine the character of normal sounds, the presence of abnormal sounds, and the regularity of cardiac rhythm. When the heart sounds are feeble or when the respiratory sounds are unduly loud, it may be necessary to have the patient hold his breath for brief intervals while we auscult. The procedure, however, if long continued brings about abnormal conditions which affect the heart, and in patients suffering from marked dyspnea, may be impossible. In deep-chested individuals it is often advantageous to have the patient lean forward while the heart is being ausculted, a procedure which brings the heart nearer to the anterior chest wall.

With the exception of the pulmonary valve, the sound produced at a given orifice is heard best, not immediately over the area beneath which the valve is situated, but a distance from it. This is because although the sound in question is still heard over its specific area, the other sounds are at that point heard less distinctly. Heart sounds and murmurs are not infrequently audible over the back. This is almost invariably the case in childhood.

THE ORIGIN AND CHARACTER OF THE HEART SOUNDS

The functional activity of the heart produces certain sounds which can be best heard over the precordium. If the bell of the stethoscope is placed over the left lower portion of the precordium (mitral area) two, occasionally three, distinct sounds will be heard. The sounds are in normal cases easily differentiable by their acoustic qualities. Under certain abnormal conditions they can be distinguished only by timing their occurrence with the cardiac impulse or if this is invisible and impalpable, by palpating the carotid pulse which occurs 0.1 second after ventricular systole. The two sounds mentioned may be imitated by the syllables, lūbb-dŭpp.

The First Sound.—The first sound is systolic in time and synchronous with ventricular contraction. It is long, more or less loud, low-pitched and terminates abruptly. It is *due to:* (*a*) muscular contraction of the ventricles; (*b*) systolic tension of the auriculo-ventricular valves; (*c*) distention of the aorta and pulmonary artery. It is loudest over the body of the heart but also heard over the base. It is normally louder as well as longer (0.8 second) than the second sound (0.05 second) from which it is separated by the short pause. Systole is shorter than diastole, therefore the first sound is preceded by the long pause.

The Second Sound.—The second sound marks the beginning of diastole, it is shorter, less loud, higher-pitched and less voluminous than the first sound. It is *due to* the sudden increase in tension of the semilunar valves. It is preceded by the short and followed by the long pause. It terminates even more abruptly than the first sound.

The Third Sound.—This can occasionally be heard especially in children, in the left lateral decubitus, and if the heart action is slow; as a faint echo of the second sound. It occurs early in diastole, about 0.1 second after the second sound, and when sufficiently marked, causes the protodiastolic gallop rhythm. It is synchronous with the early normal diastolic elevation of the apex cardiogram, and with the descending limb of the "v" wave of the jugular pulse. When, however, allowance is made for the transmission, the impulse of the protodiastolic elevation is found to be synchronous with the "h" wave. Thayer[1] believes the third sound to be *due to* the sudden tension of the mitral valve which occurs with the first inrush of blood at the beginning of diastole.

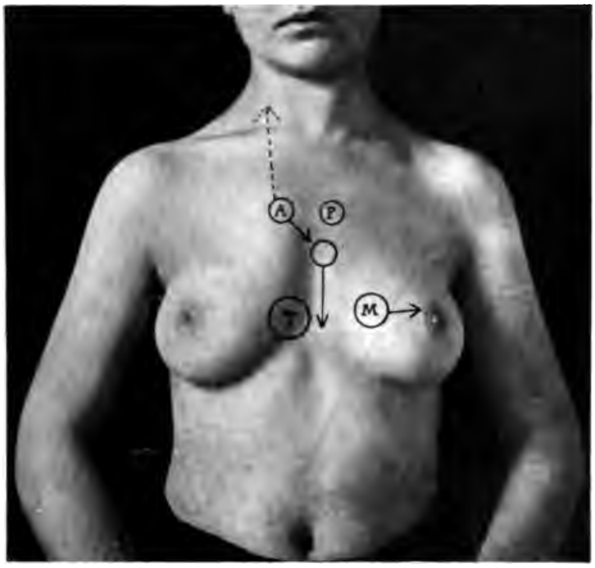

FIG. 178.—Showing the areas of the chest over which sounds produced at the various valvular orifices are generally most distinctly heard. *A*, aortic area; *P*, pulmonic area; *M*, mitral area; *T*, tricuspid area.

The unmarked area at the third left costo-sternal articulation is one at which aortic regurgitant murmur is often most loudly heard. The arrows indicate the direction of transmission of murmurs—the obstructive aortic, into the carotids, the regurgitant aortic, down the sternum and toward the apex; the regurgitant mitral, toward the axilla. (Comp. Figs. 124 and 125.)

The rhythm and accentuation of each sound varies with the location at which it is heard. At the mitral area the first sound seems louder—appears to bear the greater stress. Over the semilunar valves the accent falls upon the second sound. This is due to the variation in distance between the point of origin of the sound and the location of the stethoscopic bell.

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[1] THAYER: "Further Observation on the Third Heart Sound." *Trans. Assoc. Amer. Physicians*, May, 1909.

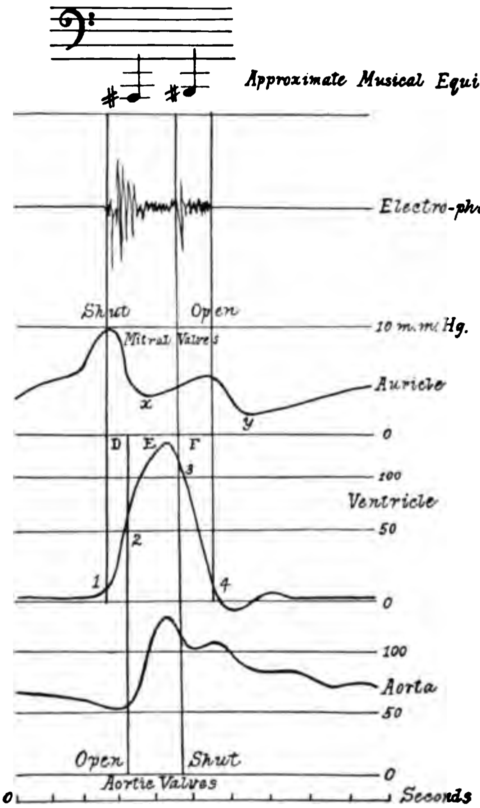

Fig. 179.—Diagrammatic representation of the heart sounds, and the pressure curves in the auricle, ventricle and aorta. *D*, presphygmic period; *E*, sphygmic period; *F*, post-sphygmic period.

The first heart sound corresponds to a vibratory rate of about 55 per second, the second sound, to a rate of about 62. The latter is, therefore, from 1 to 5 musical notes higher than the former. Both sounds are subject to considerable individual variation both as to duration and pitch, especially the second heart sound. The first sound begins from 0.01 to 0.02 seconds after the upstroke of the apical cardiogram, and ends during the fall of the level of the systolic plateau, generally during the systolic elevation of the carotid pulse. (*Gerhartz.*)

THE ACOUSTICS OF HEART SOUNDS

We speak of an heart or an arterial "tone" or sound as opposed to a murmur—a distinction made by Rouanet, and now ineradically custom sanctioned. But in a purely physical sense such a differentiation is entirely unjustified since both are the result of irregular sonorous vibrations. Indeed, of the two, the vibrations which produce a "murmur" are more rhythmic than those which produce a "sound."

Heart sounds are *produced* by vibrations of the muscular or membranous structures; not by their fluid contents. Sudden increase in valvular or arterial tension alone may produce longitudinal but not horizontal vibrations, and only the latter are capable of sound production. If an elastic band which has been stretched is plucked, horizontal vibrations are set up and a sound is produced. If the band is merely suddenly stretched by pulling upon either end, longitudinal vibrations are set up but unless accidental horizontal vibrations result from the irregularity of the jerk no sound results.

A membranous sac suddenly distended by fluid not only expands sufficiently to accommodate the increased contents, but owing to its "inertia" at first overexpands and in turn, owing to its elasticity, contracts. In this manner a series of transverse, tone-producing vibrations are set up. Thus arterial sounds are produced in all arteries in which the disturbance of equilibrium is sufficiently rapid. Sclerotic vessels are less distensible, hence the arterial sounds over such arteries often have a metallic quality owing to the very unrhythmic vibrations engendered by the stiffened tissues. Heart sounds arise in a similar manner. If, owing to valvular leakage or low arterial blood-pressure, outflow begins before a rise in tension sufficient for sonorous vibration occurs, the sounds are weak or inaudible. The first heart "sound" is only possible, therefore, if a presphygmic period of increased tension precedes the outflow of blood. The slower the pulse rate, and the lower the diastolic pressure, the easier the outflow and the weaker the first sound.

The First Heart Sound.—The first heart sound is mainly due to vibrations of heart walls and of the valves. The latter elements contribute in no small measure to the total sound. (The intra-auricular pressure is so low compared to that of the ventricles, that the cuspid valves close quickly, and owing to their anatomical configuration have a large vibratory excursion.)

Following the muscular and valvular elements of the first sound by 0.06 to 0.07 second (presphygmic period) the vascular element due to the sudden distention of the aorta and the pulmonary artery occurs. Ordinarily these three elements are fused into a single sound, but if the presphygmic period is delayed the different sound elements become asynchronous and a recognizable *splitting of the first sound* occurs.

The Second Heart Sound.—The second heart sound is due to sudden increase of tension and subsequent vibration of the aortic and pulmonary valve during closure. The difference in intra-ventricular and intra-arterial pressure at the end of systole being great, especially in the former case, the valves snap back, and vibrate until equilibrium is reëstablished. The sounds produced by aortic and pulmonic closure while not quite synchronous, are separated normally by too short a time interval to be auditorily appreciable as separate sounds. When pathologically the normal pressure differences are exaggerated, a *split, second sound* is noted, a condition which is more readily appreciable if the first element is the weaker of the two.

Inasmuch as our auditory impressions of the two elements of the second sound are always to some extent fused, it is probable that we can recognize one as increased over the other only when at least a 2 to 1 difference in intensity prevails. When a "marked increase" both of pitch and intensity of the pulmonic second sound is noted, the actual relative intensity is several times greater. In attempting to estimate relative differences in intensity auscultation should be practised close to the sternal margin and not at a distance from it, since the relative difference tends to become less marked in the process of conduction (Geigel).

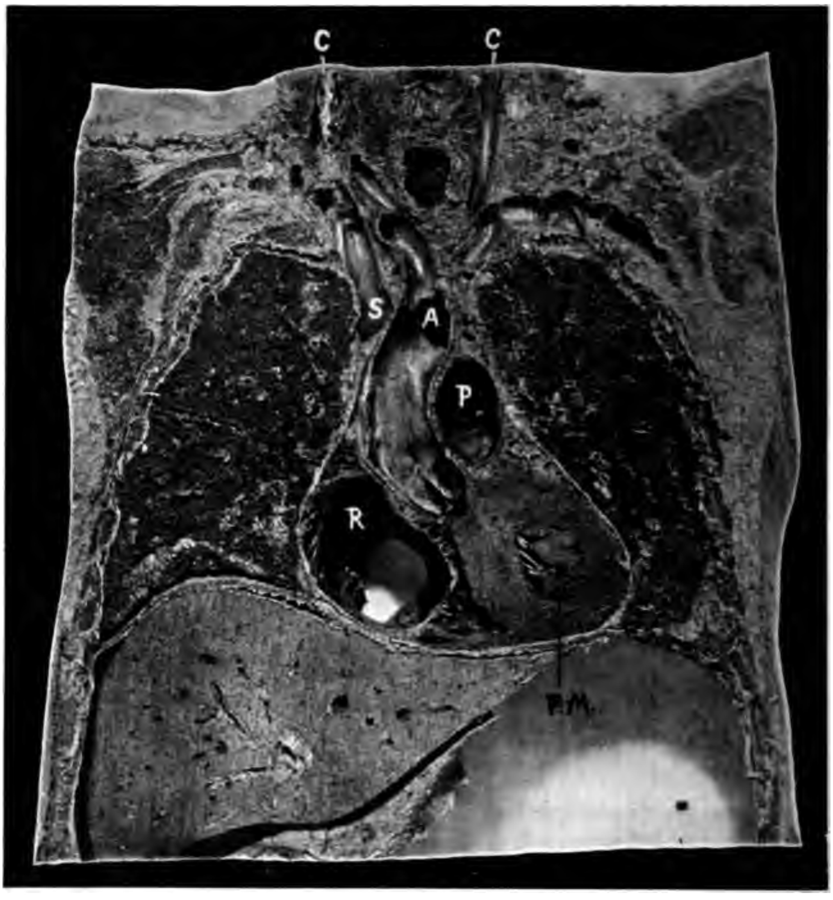

FIG. 180.—SECTION OF THE THORAX VIEWED FROM IN FRONT. Systolic (obstructive) aortic murmurs are transmitted in the direction of blood flow, upward into the carotid arteries. Systolic (regurgitant) mitral murmurs are transmitted in the opposite direction to the blood current, toward the left axilla, being conducted thence by the chordæ tendineæ and the papillary muscles. *A* = aorta; *P* = pulmonary artery; *C* = carotid arteries; *P.M.* = papillary muscle; *R* = right auricle; *S* = superior vena cava.

THE INDIVIDUAL VARIATION OF HEART SOUNDS

There is no absolutely normal standard of the heart sounds and a certain allowance must be made for individual peculiarities. As a general rule such variations can be explained either upon anatomic grounds—the size of the lungs, the depth of the chest, etc., or upon the basis of age and sex.

Substernal Sounds.—In a considerable number of perfectly healthy individuals the heart sounds heard just over the ensiform cartilage, and in its immediate vicinity, have a peculiar harsh, scratching, scraping or crunching quality closely resembling a pericardial friction sound. This sound which has been described as the xypho-sternal crunch, seems superficial, is increased and sometimes audible only when the patient leans forward or to the left. It is short and heard with variable intensity during both systole and diastole. Its etiology is uncertain. Blumer has suggested that it is due to changes in tension of the loose areolar tissue in the sterno-pericardial ligament. This sound is frequently met in cases of cardioptosis. It is a relatively frequent normal phenomenon, the importance of which lies in the fact that it may readily be mistaken for a pericardial friction, or an obstructive mitral murmur.

The Presystolic Sound.—The recent electrophonographic studies of Bridgman have corroborated by means of graphic records, the existence of a normal presystolic sound. The sound in question, while clinically unusual, has long been recognized as a diagnostic stumbling block, since it may closely simulate the faint presystolic murmur of a slight degree of mitral stenosis.

This sound is perhaps due to the tension of the ventricular walls, and the fact that it is not more frequently heard appears to be owing to the fact that the sound waves are normally below the limits of human audibility.

DISPROPORTIONATE INTENSITY OF THE HEART SOUNDS

The relative intensity of normal heart sounds as determined with the Oertel stethoscope by Bock are: mitral sound, 40; pulmonic sound, 18; aortic sound, 20. The sound heard over the mitral area is normally twice as loud as the aortic.

Accentuation of the first sound is due to increased contractile force of the papillary and ventricular muscles. It occurs after exertion, during overaction (mental excitement, the early stages of fevers, etc.) and in ventricular hypertrophy.

The first heart sound is diminished in intensity, assumes the quality of the second sound, in protracted fevers (typhoid, etc.), in some valvular diseases, especially in mitral insufficiency, and in fatty infiltration or degeneration of the myocardium. The muscular element becomes diminished and it assumes a "valvular" quality, owing to the preponderance of its second sound component, *i.e.*, the snapping back of the mitral valve.

Accentuation of the second sound indicates increased rapidity of closure, and this in turn relatively increased vascular pressure, in the aorta or pulmonary artery. The sound is due not to the mechanical act of valvular closure, but to sudden increase in tension and subsequent vibration of the valves.

The pulmonic second sound is normally louder up to twenty-five or thirty years of age, after which the aortic tends to become louder. When auscultation is practised (experimentally) directly over the vessels, the aortic sound is the louder. In 90 per cent. of all children under ten years of age, the pulmonic sound is the louder (the artery is more superficial), and in about 10 per cent. of the cases a splitting of the second sound can be recognized.

The *second pulmonic sound is louder in the recumbent posture,* and in conditions which increase the pressure in the lesser circulation—pulmonary consolidation, pleural effusion, mitral disease, emphysema—provided the right heart is sufficiently strong to maintain the increased tension, *i.e.*, the tricuspid valve remains competent. *When, therefore, we say that the second sound is accentuated we mean that it is relatively louder than it should be in relation to age and posture.* This phenomenon indicates increased pressure in the lesser circulation.

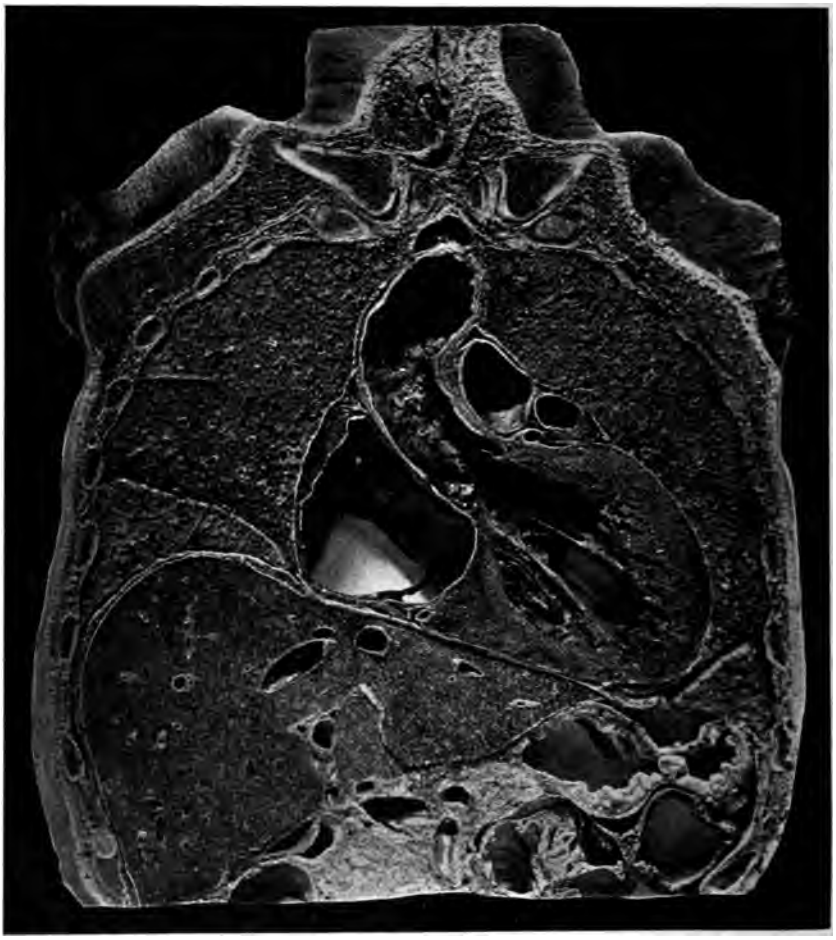

Fig. 181.—Left ventricular hypertrophy. Extreme hypertrophy of the heart, especially of the left ventricle, occurs most characteristically in aortic valvular disease, or as a result of long-standing arterial hypertension. It is clinically characterized by 1. a forcible, heaving cardiac impulse; 2. displacement of the apex impulse downward and to the left; 3. a long, low-pitched, booming first sound; and 4. unless the aortic valves are insufficient, or myocardial weakness pronounced, a clear, high-pitched, ringing accentuation of the second sound both at the apex and at the aortic area.

The *aortic second sound is increased* in arterial hypertension, provided the ventricular strength be sufficient. This occurs especially in glomerulo-nephritis, often in arterio-sclerosis and in pregnancy (increased mass of blood or blood-pressure). It is always louder than the pulmonic sound after the sixth decade. Accentuation of the aortic second sound can be demonstrated in about two-thirds of the cases of arterial hypertension. The fact that it is not demonstrable in all the cases may be due to the presence of obesity, pulmonary emphysema, etc. Occasionally a clear, loud metallic sound is heard in cases of syphilitic aortitis even with a normal blood-pressure, showing that arterial hypertension is not the only determining factor. The aortic second sound is *diminished* in intensity in arterial hypotension and in conditions associated with imperfect filling of the aorta—mitral or aortic obstruction, exhausting diarrhea, and when the semilunar valves have lost their elasticity.

CHANGES IN THE PITCH OF HEART SOUNDS

Increase in the pitch of the first sound occurs when contraction is rapid; increased pitch of the second sound occurs when the semilunar valves close under higher tension. When marked, the latter imparts a ringing, high-pitched, metallic quality to the second sound which is quite as indicative of increased tension as is the actual intensity of the sound. When air-containing cavities exist near the heart (pneumothorax, pulmonary tuberculosis, tympanitis, etc.) the heart sounds may also take on a ringing, metallic quality owing to the resonating properties of the neighboring cavities.

In case of rapid heart action and low blood-pressure—fevers, muscular exertion, etc.—the first sound is shorter, more snappy and higher in pitch. In cardiac hypertrophy with increased blood-pressure it is more booming and lower in pitch. In cardiac dilatation it is short, sharp and flapping. A weak first sound may be due not to lack of force but to slowness of muscular contraction. The quicker the contraction and the more sudden its termination, the louder the sound (Krehl). In children the first sound is more snappy and high-pitched, owing to a preponderance of the valvular sound components.

THE REDUPLICATION OF HEART SOUNDS

Both heart sounds are composite. If any one of the component elements falls out of time a reduplication occurs, two sounds instead of one being heard. If the time interval between the component sound elements is a short one we speak of a splitting of the sound; if more prolonged, of a reduplication. The introduction of some abnormal element may also cause a reduplication. Instead of the normal lub-dupp we hear tu-rub-b-lupp.

Reduplication of the first sound which is most commonly heard at the apex, and in the erect posture, is probably in the main due to delayed contraction of the papillary muscles. These structures being supplied by terminal arteries, are readily affected by fatigue and by noxious influences. Reduplication may, however, be due to a late production of the vascular element (expulsion tone), especially if the presphygmic period is prolonged. It is often heard in children, in thin-chested individuals, and pathologically in arterio-sclerosis, etc.

Reduplication of the second sound may be due to: (*a*) abnormal pressure relations and markedly asynchronous closure of the aortic and pulmonic valves; (*b*) conditions hastening or preventing a sudden increase in tension of the semilunar leaflets—stiff valves.

Example.—A deep inspiration increases the blood in the lungs, and decreases the amount in the left ventricle. The pressure in the aorta, therefore, increases disproportionately to that in the left ventricle and the valves close more quickly. This also occurs in mitral stenosis, whereas the condition is reversed in mitral insufficiency. Splitting of the first sound yields an anapestic rhythm (intervene) ⏑⏑—. Splitting of the second sound yields a dactylic rhythm (merciful)—⏑⏑ (Cabot).

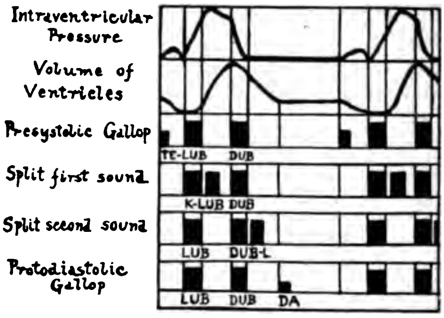

FIG. 182.—Illustrating split heart sounds, gallop rhythms and their phonetic equivalent. (*After Hirschfelder.*)

Abnormal accentuation of the aortic second sound indicates increased arterial pressure; increased intensity and pitch of the pulmonic second sound points to increased pressure in the lesser circulation.

CHANGES IN THE RHYTHM OF THE HEART SOUNDS

When from any cause the heart is weakened and the diastolic pause shortened, the heart sounds tend to become equally spaced and valvular in character. This condition is known as "*pendular rhythm*" owing to its resemblance to a ticking clock. It is generally due to prolongation of ventricular systole, and hence to a delay in the appearance of the second sound. When this pendular rhythm is associated with a rapid pulse a disproportionate shortening of diastole occurs, and the rhythm is spoken of as *embryocardia* (fetal rhythm). It is generally associated with a monocrotic pulse and heard in cases of cardiac dilatation.

The Presystolic Gallop Rhythm (⏑—⏑) " symbolic."—In this condition a third sound is introduced into the cardiac cycle which occurs just before ventricular systole. It takes its name from its similarity to the footfall of a galloping horse, and is generally due to abnormally and audibly, asynchronous contraction of the right and the left ventricles—the former preceding. It is sometimes due to abnormally loud auricular contraction and hence may occur when these chambers are hypertrophied as in the early stages of mitral obstruction. It occurs chiefly in cases of arterial hypertension associated with a dilating hypertrophy. Electrocardiographically it is almost invariably characterized by a split "R" wave. Presystolic gallop rhythm may also occur in the course of acute infections, especially diphtheria, scarlatina and rheumatic fever. It is a tress signal from the heart and prognosis is worse if it occurs with o- than hypertension.

The Protodiastolic Gallop Rhythm (◡◡—) "intervene."—This is iply an exaggeration of the normal third sound and is due to the lden upward snap of the auriculo-ventricular valves early in diastole. is generally best heard at the base of the heart. It occurs especially en the rate is slow and corresponds to the appearance of the "h" wave the phlebogram. It is heard especially in mitral obstruction, aortic ufficiency, and adhesive pericarditis.

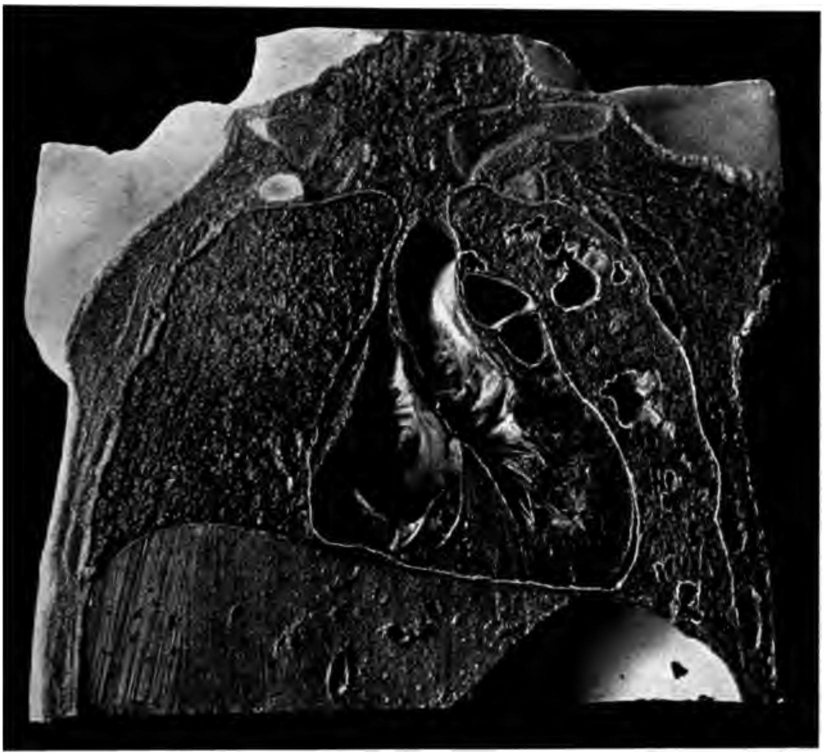

FIG. 183.—*Advanced pulmonary tuberculosis of the left lung* with retraction, consolida- ı, cavitation and cardiac displacement of an hypertrophied heart to the left. In such s the heart sounds are often clearly heard over the whole affected side, owing to di- ished reflection at the heart- (solid) lung junction and to the resonating properties of the ities. Furthermore, the lung is generally contracted exposing a large cardiac surface to chest wall. Still another factor is the concomitant emaciation of the patient resulting iminished superficial tissue. Conduction of the heart sounds to the opposite side of the st in right-sided pulmonary tuberculosis was pointed out by J. M. DaCosta and is some- es described as "DaCosta's sign."

Occasionally the sound may be heard in mid-diastole, owing to the t that the auricles begin to contract at this time. This generally indi- es a high grade of cardiac asthenia, as in very toxic and prolonged es of typhoid fever.

CHAPTER XX

HEART MURMURS

Acoustics.—Heart murmurs are abnormal sounds, having generally a blowing quality, which is due to more or less rhythmic vibrations of the cardiac or arterial tissues. They are generally produced at or near a valvular orifice by abnormalities in contour or structure. Such conditions are brought about (1) either by *organic changes* which result in thickening, stiffening, roughening, constriction, dilatation, perforation or retraction of the valvular tissues or (2) by *functional lesions* resulting in loss of muscular tonus. The latter occur chiefly at the mitral and tricuspid orifices. *Heart murmurs may be produced* (1) when blood flows from a cavity into a cylinder; (2) when blood flows from a cylinder into a cavity; (3) when membranes vibrate in the blood stream; (4) when the endocardium or intima are roughened.

"Whatever contracts an orifice, whatever dilates a cavity, whatever establishes an orifice or a cavity where none should be, will disturb the even flow of blood, and produce vibration and a murmur" (Gee). The *heart sound* consists of a single, intense, demarked auditory impression. A *heart murmur* gives us the effect of unevenly composed sounds which arise from rapidly varying, irregular, sound production. The heart sound has been compared to a single stroke upon a drum; the murmur, to the sound produced by blowing into a pipe. The sound bears the same relation to the murmur as does a pistol shot, to the surge of the sea. *The difference, therefore, consists in the suddenness of onset and of ending of the sound, as compared to the gradual beginning and uncertain termination of the murmur.*

The fact that heart murmurs are produced by vibrations of the tissues and not by the vibrations of the blood stream is shown by the following facts.

I. If by way of experiment (Fig. 184) we introduce a flowing stream of water by means of a tube into a beaker and listen with a stethoscope, (1) above the fluid level: no sound is heard. (2) If the bell be immersed, the tube mouth being at "A": a murmur is heard at "C." (3) If the tube be raised: the murmur becomes feebler, even when the current surrounds the bell, showing that the sound waves are not produced by the water directly, but by the vibrations imparted to the wall of the beaker. The sounds increase in intensity, the nearer the tube to the wall, *i.e.*, the larger the surface contact between the glass and the fluid vein. On withdrawal of the tube all sound ceases, despite the fact that the current still persists for a time. That this cessation is not due merely to an inadequately rapid current is shown by the fact that pouring H_2SO_4 into water, which owing to greater specific gravity and the generation of heat must cause much more rapid currents, does not alter the results.

II. Further, it can be shown mathematically that the sounds produced in the heart and blood-vessels are far too low in pitch to be due to sonorous vibrations produced in chambers of such small dimensions.

The first heart sound has been shown by Gerhardt and Funke, to correspond to 198 vibrations per second. To produce by fluid vibrations a sound of a corresponding pitch to that of the heart, would, according to Helmholtz's formula call for a ventricle with a capacity of about 24 liters! Although these figures are doubtless high owing to the fact that Helmholtz's formula presupposes a cavity communicating with the atmosphere, yet even allowing liberally for such facts, it would be quite impossible to produce sounds similar to those of the heart or of murmurs by vibrations of the blood columns in the heart and great vessels.

Unquestionably then, *heart sounds and murmurs must be due to horizontal vibrations of the heart and vessel walls* (Williams, Kiwisch, Weber). *The heart walls and* [*the blood stream play the respective parts of the violin string and the bow* (Weber).

The mechanism by which mural vibrations are produced is as follows:

I. In flowing from "A" to "B" (Fig. 185) the fluid particles deviate from their linear course and produce a series of impactions on the walls of "B," because the adhesion between the wall and the outer fluid layer is greater than the cohesion between the fluid particles. This tends to create a vacuum at "C" which in turn causes the elastic walls of "B" to fall inward. Such a movement temporarily overcomes the vacuum, thus again allowing the walls to regain their original position. A regularly recurring series of such events sets up continuous lateral vibrations of the vascular walls in the expanded section ("B"). In the constricted portion ("A") a series of vibrations is established by alternate suction and compression engendered in "B." These vibratory waves have "D" as their nodal point. Rapidity of flow greatly favors murmur production. Clinically the production of murmurs through expansion of the blood channels finds many exemplifications—valvular lesions, aneurisms, etc.

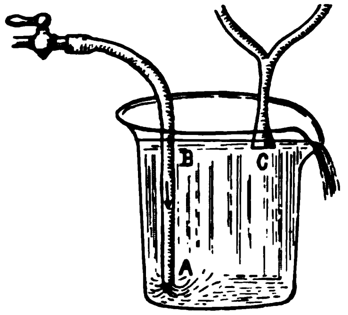

FIG. 184.

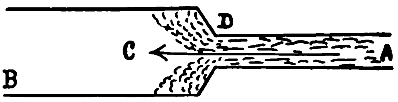

FIG. 185.

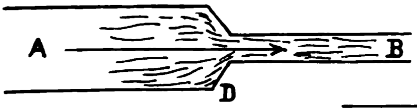

FIG. 186.

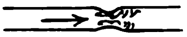

FIG. 186a.

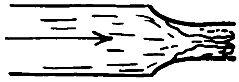

FIG. 187.

II. The reverse condition, in which there is a sudden contraction of the blood channels, may also produce murmurs (Fig. 186).

Here the constriction at "D" raises the pressure. This tends to force the stenotic opening apart. When this has been accomplished pressure temporarily falls again. Such alterations of pressure throw the vascular walls in both sections "A" and "B" into alternate transverse vibration. The ideal conditions for the production of a murmur exist in case of fluid flowing through a constriction (Fig. 186a).

III. Membranous diaphragms, *e.g.*, heart valves or vegetations in the blood stream, may, by flapping like a sail in the wind, be thrown into vibration, and may be a further cause in the production of murmurs (Fig. 187). (Compare Fig. 212.)

IV. With sufficient rapidity of the blood current, even smooth-walled vessels of uniform caliber may be thrown into vibration (this has been demonstrated by Weber), for no vessel is absolutely smooth; and slight degrees of roughening are equivalent to minute stenoses. Only in this sense can murmurs be said to result from "friction of the blood stream" (Geigel). "Fluid of any kind, flowing at any speed through a cylindrical tube, will not cause a murmur, even if the tube be curved, so long as it retains its cylindrical form" (McLennan).

THE TECHNIQUE OF CARDIAC AUSCULTATION

I. Place the bell of the stethoscope firmly in the fourth interspace or over the fifth rib just within the cardiac impulse. Ignore the respiratory sounds and determine which is the first sound. This is accomplished by observing (1) that it is synchronous with the cardiac impulse; (2) that it follows the long pause; (3) that it is a lower pitch as well as longer and louder than the second sound. The last criterion is, of course, not always reliable.

Note its : (1) rate, (2) rhythm, (3) intensity, (4) quality, (5) duration, (6) pitch and (7) mode of beginning and of termination.

II. Examine the sounds heard over the aortic, pulmonic and tricuspid areas in a similar manner (Fig. 187).

VARIATIONS IN THE INTENSITY OF HEART SOUNDS

Considerable individual variation in the loudness of heart sounds exists. The sounds are:

(a) **Decreased** in recumbency, in adiposity, in men with great muscular development, in women with large mammæ, in anasarca, in pericardial or left-sided pleural effusions, in pulmonary emphysema, in myocardial weakness and in the agonal period. Faint irregular, incoördinated sounds are sometimes heard for a time after death and probably result from auricular contractions, since the right auricle is the last part of the heart to die. The heart sounds may be very faint and muffled even in perfect health.

(b) **Increased,** during cardiac overaction—physical exertion, mental excitement; if the lungs are small, or if they are retracted, or consolidated in the neighborhood of the heart; or if the heart is hypertrophied.

Occasionally the heart sounds can be heard all over the chest. *Changes in the relative intensity of the first and second sounds are much more important than mere changes in the actual intensity of both sounds.*

Certain *facts have been demonstrated experimentally which have practical clinical significance.* (1) A murmur is more easily produced, if the walls of the tube be thin, if the inner surface be rough, if the tube be rigid, and if viscosity is increased. (2) Increased tension has very little effect provided the rate remains the same. (3) Increasing the compression of the tube will increase the murmur up to a maximum, after which it tends to disappear. (4) With a certain grade of rapidity and compression, the murmur becomes finer and more musical (T. Weber).

From the standpoint of physics no sharp distinction can be drawn between heart sounds and murmurs because physically speaking both are murmurs. *Heart sounds are due to a single impulse giving rise to vibrations of the cardiac or vascular wall; a murmur is due to repeated vibratory impulses.* The difference is similar to that of plucking or stroking a violin string. Hence sounds are generally shorter in duration, but this is not an essential difference: a murmur may be short and a sound long (for example, the sound of a kettle drum and a tuning fork, although each is produced by a single blow). The real difference lies in the fact that *in the case of the sound, the first vibration is the greatest, whereas in the case of the murmur, we have at the outset for a time, a continuous series of equal vibratory excursions.*

Impure sounds are a mixture of heart sound and heart murmur with no very definite preponderance of either. Thus in a given case one observer would say "the first sound is murmurish, impure," while another might with equal justice and correctness say there is "a faint, short systolic murmur." Impure sounds are occasionally heard in health, but indicate a functionally if not structurally imperfect valve (roughness, relaxation or stiffness). Under exercise they often develop into definite murmurs. Sounds are to a certain extent conducted along the arterial wall, but probably not to any great degree when the vessels are surrounded by good conducting media. There is also some conduction by the fluid itself.

ENDOCARDIAL MURMURS

Heart murmurs may be caused by perforation, constriction, dilatation, or roughening of the blood channels, especially the valvular orifices. The lesions furnishing these conditions may be:

1. **Organic.**
   (a) *Obstruction* to the onward flow of blood—stenosis.
   (b) Valvular *insufficiency*, allowing an escape of blood backward—regurgitation, incompetency.
2. **Functional** (without structural alterations) insufficiency of the:
   (a) *Semilunar valves*—increased arterial pressure and dilatation of the aortic or pulmonic rings.
   (b) *Cuspid valves*—relaxation of the mitral or tricuspid sphincters, or improper functionation of the papillary muscles.

Relative insufficiency is much more common in case of the cuspid than in that of the semilunar valves. Of the former, the tricuspid is structurally a much less perfect valve than is the mitral, and hence is much more prone to leak under conditions of strain, in which case it acts to a certain extent as a safety valve to the right heart, with a second line of defense in the not far distant liver, against which the blood thrust of the right heart is delivered.

The murmur of a stenosis is produced by a current travelling the normal direction, and is sometimes spoken of as an *onward murmur.* It occurs at the time when the valve should be open.

The murmur of an insufficiency is produced by a reflux of blood and occurs at a time when the valve should be closed—*backward murmur.*

THE TIME RELATIONS OF VALVULAR MURMURS

	Systolic	Diastolic
....	Mitral Tricuspid	Aorta Pulmonary artery
....	Aorta Pulmonary artery	(Presystolic) Mitral Tricuspid

The Differentiation of Valvular Murmurs.—Several murmurs are often coincidently present in a given case and on the other hand the same murmur may be heard over different areas of the precordium. In such cases *we judge of the identity or non-identity of such murmurs by the following criteria*: (1) location, (2) time, (3) transmission, (4) quality, (5) pitch, (6) duration, (7) intensity. Among these, *place, time, and direction of conduction are by far the most important.*

The Location of Murmurs.—A murmur is generally best heard at the point of the chest wall which is nearest to the orifice at which it is produced. The location at which the different valvular sounds are best heard has already been considered (p. 217). The same rules govern the audibility of murmurs.

The Time of Murmurs.—It is essential to determine the exact time and duration of a murmur in its relation to the cardiac cycle—whether it occurs in systole, diastole or presystole. This is accomplished by noting the relation of the murmur to the (1) apex impulse, (2) first sound, (3) long pause. The terms *protosystolic, mesosystolic* and *telesystolic* are applied to murmurs which occupy respectively only the beginning, middle or end of systole.

The Transmission of Murmurs.—Murmurs are transmitted, that is they are heard at certain areas of the periphery other than those overlying the location of their origin, or of their normally greatest intensity. Murmurs are conducted to different regions generally in the direction of the blood current, just "as wind carries sound." Murmurs are, however, not merely conducted to the surface by the nearest possible route. The sound-carrying quality of the neighboring tissues also plays a most important rôle. Thus the murmur of aortic insufficiency may be loudest over the ensiform cartilage whence it is conducted from the second right interspace; whereas the mitral regurgitant murmur, produced deep within the mediastinum, is conducted in an opposite direction to that of the blood flow, by means of the papillary muscles, to the region of the cardiac apex, the axilla, or the scapular angle.

The Quality of Murmurs.—Murmurs vary in quality and are hence described as being musical, non-musical; harsh, soft; blowing, scraping; squeaking, etc. The pitch is also of great importance. These facts are significant since they aid one in distinguishing between murmurs when two or more are present. It is to be remembered, however, that a murmur may undergo modification dependent upon the point at which one auscults; and further, that the same murmur may change very considerably in quality as a result of numerous conditions such as myocardial weakness, exercise, posture, blood-pressure, etc. *One cannot judge of the type or seriousness of a lesion by the quality or intensity of the murmur.* Indeed, a loud murmur may denote a good heart muscle and a weak murmur, myocardial weakness. When the latter is marked an organic murmur previously present may disappear entirely to reappear when compensation improves. As a general rule, however, musical and scratchy endocardial murmurs are rarely functional. When an organic lesion is progressive, the murmur increases in intensity up to a certain point, beyond which it recedes, either as the result of muscular weakness or on account of the extent of the valvular damage. Systolic murmurs are generally louder than diastolic murmurs, especially in recumbency. On account of their faintness and low pitch, diastolic murmurs are easily overlooked. Systolic murmurs may be present when the patient lies down and may disappear when he is erect. Obstructive murmurs are generally louder in the erect posture. Aortic and pulmonic murmurs are less affected by posture than those produced at the mitral or tricuspid orifices. Most murmurs are louder after exertion, and some may require physical exercise to make them audible. Hemic murmurs resulting either from anemia alone, or from anemia together with a dilatation of the pulmonary artery, are best heard at the pulmonic area. They are soft and blowing, low in pitch, variable in character, indefinite in transmission. Murmurs are also described as being *crescendo* or *diminuendo* in character, the terms being used in the musical sense to indicate a continuous increase or decrease respectively of their intensity. The murmur of mitral stenosis is often typically crescendo, that of aortic insufficiency often diminuendo, in quality.

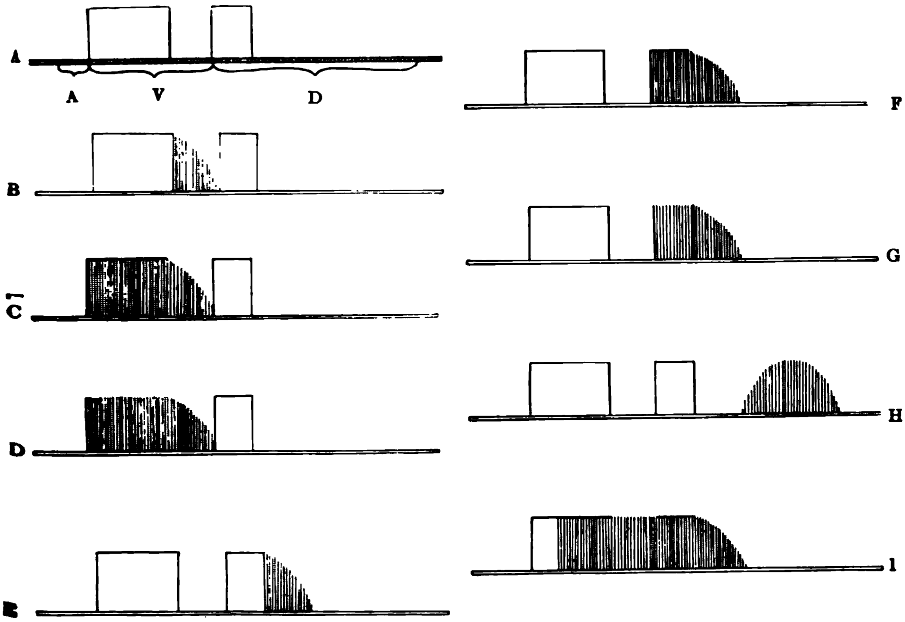

FIG. 188.—DIAGRAM ILLUSTRATING HEART SOUNDS AND MURMURS.

A: Normal heart sounds. *A*, auricular systole; *V*, ventricular systole; *D*, ventricular diastole. The sounds begin and end abruptly.

B: Systolic murmur following first sound. The first sound terminates gradually.

C: Systolic murmur accompanying first sound. The first sound is "impure" and fades away gradually.

D: Systolic murmur replacing first sound. The first sound is supplanted by a blowing sound.

E: Diastolic murmur following the second sound. The second sound is followed by a blowing sound.

F: Diastolic murmur accompanying the second sound. The second sound fades away gradually.

G: Diastolic murmur replacing the second sound. Instead of a clear-cut second sound one hears a blowing sound. This is often low pitched and heard with difficulty. The absence of the second sound always suggests careful investigation.

H: Late diastolic murmur.

I: Continuous systolic and diastolic murmur in patent ductus arteriosus.

Even organic murmurs may be variable in quality and intensity to the point of actual disappearance, either constantly or intermittently in a series of successive cardiac cycles. Such variation is not uncommon in auricular fibrillation, extrasystole and heart block owing to variable degrees of auriculo-ventricular coördination and rate of blood flow. With the onset of auricular fibrillation or of paroxysmal tachycardia, murmurs often disappear entirely, to return when the normal rhythm is reëstablished.

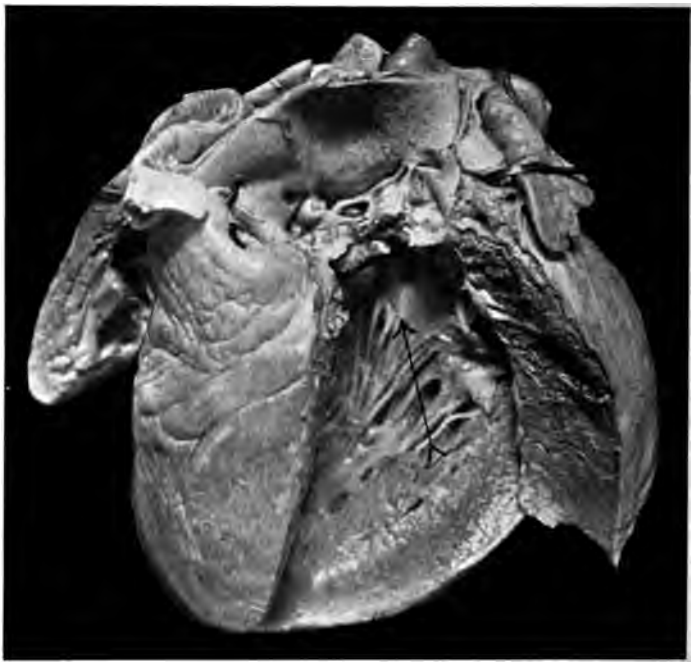

FIG. 189.—AORTIC OBSTRUCTION. The aortic leaflets are fused together as the result of inflammatory adhesion, leaving only a small elliptical opening. The arrow indicates the direction of blood flow. The physical signs of aortic obstruction are a systolic thrill and a loud crescendo murmur at the base of the heart, the latter being transmitted into the carotid arteries. The pulse is small in volume and the systolic plateau shows a long, slow, gradual ascent and descent.

The left ventricle becomes greatly hypertrophied and hence the cardiac impulse is displaced downward and outward, and the longitudinal diameter of the heart is increased. When, as is usually the case, mitral insufficiency exists, the vertical diameter is also increased. (See Fig. 172.)

INDIVIDUAL VALVULAR MURMURS

1. **The systolic aortic murmur** results from roughening or obstruction at the aortic orifice, or dilatation of the aorta. It is best heard at the second or third left intercostal space and is transmitted into the carotid arteries. Phonetic equivalent: Lu-f-f-f-Dupp.

2. **The diastolic aortic murmur** results from insufficiency, retraction, separation (vegetations) or perforation of the aortic leaflets. It is best heard at second right or fourth left intercostal space, close to the sternum, and is transmitted toward the apex and to the ensiform cartilage. It is sometimes clearly audible over the spinous processes of the first and

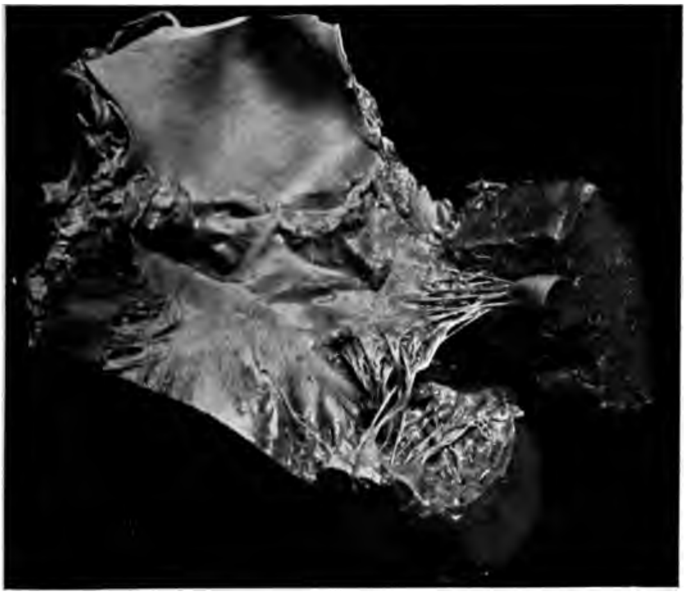

Fig. 190.—Aortic roughening and calcification. The aortic valves are stiffened and careous, the mouths of the coronary arteries are involved in the arterio-sclerotic process. e left ventricles and papillary muscles are hypertrophied.

Physical signs. A systolic aortic murmur transmitted into the carotid arteries. A cible heaving, cardiac impulse displaced downward and to the left. A loud booming first ind; a clear-cut, ringing, high-pitched, accentuated aortic second sound. This lesion is ry common in advanced years and is *often erroneously diagnosticated as aortic obstruction.*

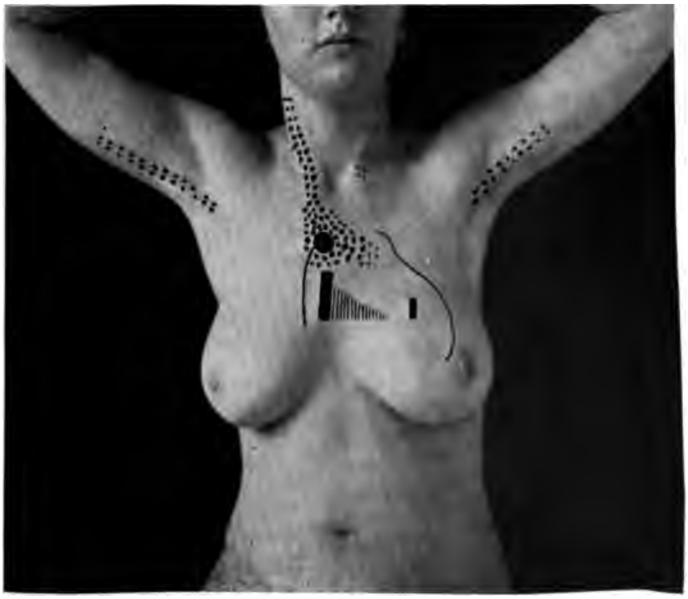

Fig. 191.—Aortic obstruction. The dotted areas indicate the locations over which the stolic aortic murmur may be heard. On the precordium has been drawn a diagram to ustrate the relationship of the murmur to the heart sounds.

second thoracic vertebræ. Phonetic equivalent: Lub Duf-f- 417).

3. **The systolic mitral murmur** is due to valvular insufficiency may result from retraction, perforation or vegetations upon the v curtains; also from shortening of the chordæ tendineæ; or from dil of the mitral sphincter. It is best heard at the mitral area and ducted toward the left axilla and sometimes to the angle of t

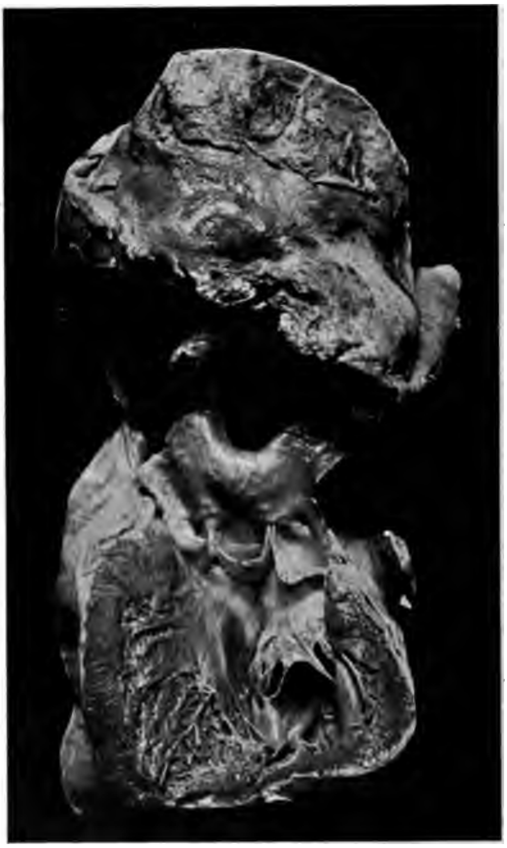

Fig. 192.—Aortic aneurism. Showing a heart with the left ventricle exp aortic valves, and beyond them a large aortic aneurism. Such a sudden wideni blood channel (aorta) sets up eddies in the blood stream and will cause a systolic n the current is sufficiently rapid and if the aneurism is not filled with a blood clot. cases of aneurism the aortic valves are incompetent, owing to dilatation of the aor and thus a diastolic (regurgitant) murmur is superadded. The double and mo continuous murmur with a rhythmic accentuation due to systole is known as a *br*

scapula. The former is in reverse of the direction of the blood and is mainly due to conduction by means of the papillary n "The chordæ tendineæ transmit the mitral murmur from the mit ments to those portions of the heart into which they are inserted." murmur heard in the left axilla in mitral insufficiency is chiefly

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[1] De Sautelle and Grey: *Arch. Int. Med.*, December, 1911.

the proximity of the insertion of the anterior papillary muscle and depends only in small part upon conduction by means of the ventricular wall. The experimental transplantation of a papillary muscle alters the direction of murmur transmission. The posterior papillary muscle often conducts the mitral regurgitant murmur to the scapular angle. At times the murmur produced at an incompetent mitral orifice is heard over the dilated left auricular appendage—in the third left intercostal space. Phonetic equivalent: Luf-f-f Dúpp.

4. **The presystolic mitral murmur** is the result of mitral obstruction. It is heard best at or just within the apex. Often one of its chief characteristics is its *crescendo quality*, which has been ascribed to (*a*) powerful diastolic ventricular suction, (*b*) to auricular systole, (*c*) to increased pressure in the pulmonary circulation. It is often associated with a snappy, high-pitched first sound. It is rough and rumbling in character, and often followed by a systolic murmur—because a valve which is sufficiently indurated to produce an obstruction is generally incapable of complete systolic closure (insufficiency). It is generally accompanied by a fine presystolic thrill, and if the right heart is competent by an

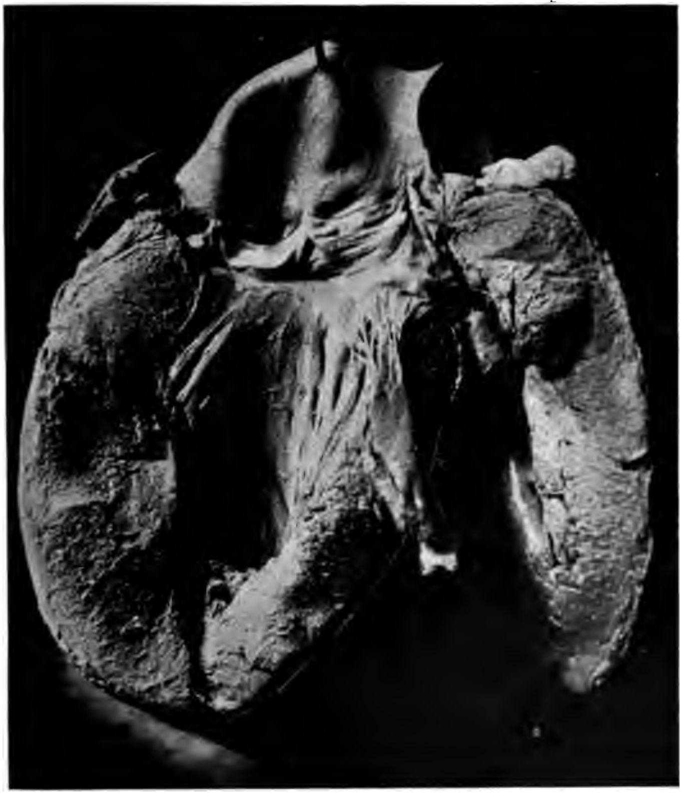


FIG. 193.—CARDIAC HYPERTROPHY. The *cor bovinum* occurs characteristically in aortic disease. It is clinically manifested by: displacement of the cardiac impulse, downward and to the left; a forcible heaving impulse, and a loud, low-pitched booming first sound. It generally results from aortic insufficiency beginning in early life.

accentuated pulmonic second sound. The murmur is most intense when blood flow is most rapid—when the differential pressure between auricle and ventricle is greatest. Hence, *the murmur of mitral obstruction may occupy any part of the diastolic interval.* Thus with a slowly beating heart and a powerful auricle the murmur appears in presystole. If, however, stenosis is marked and the pulse rate rapid, "an early diastolic rumble may be added; the period of silence between the two murmurs corresponds to the period when filling is at its slowest" (Lewis). The intensity of the murmur is in most instances increased by exercise or the recumbent posture, which factors increase the cardiac output.

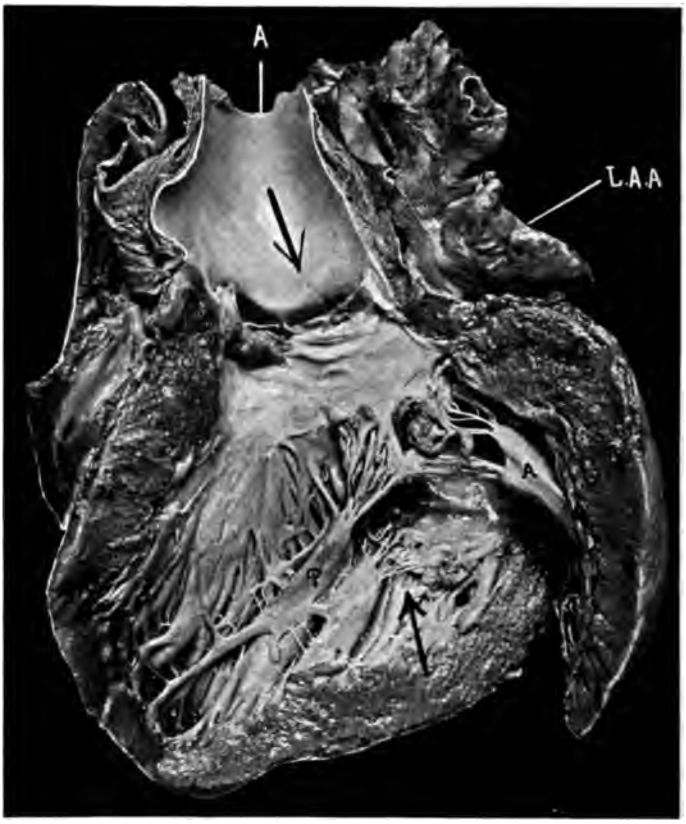
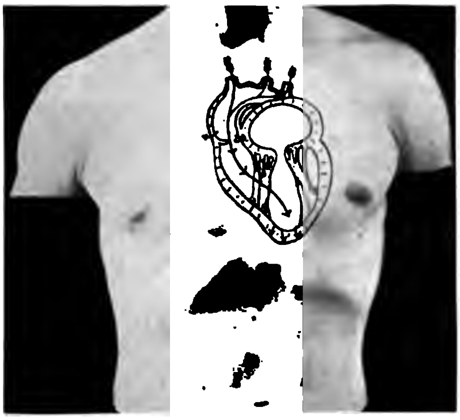


FIG. 194.—AORTIC AND MITRAL INSUFFICIENCY DUE TO ACUTE ENDOCARDITIS. Fresh vegetations are seen on the respective valves. The heart is hypertrophied, the aorta normal. The arrows indicate the direction of the regurgitant blood stream. The murmur is transmitted from the aortic valves downward toward the apex, in the direction of the blood flow. In the case of the mitral valve it is conducted in a reverse direction to that of blood flow, toward the axilla and the angle of the scapula, by means of the papillary muscles. *A*, anterior and *P*, posterior, papillary muscle. *L.A.A.*, left auricular appendix.

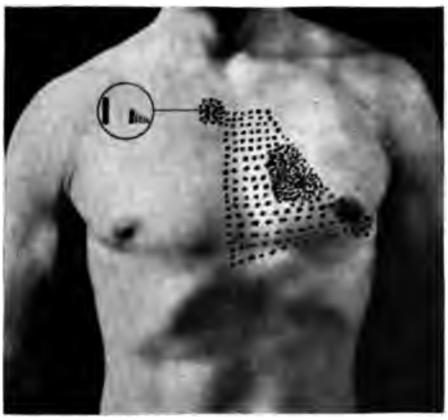
Physical signs. A heaving impulse, displaced downward and to the left, pulsating carotid arteries, a water-hammer pulse. Cardiac dulness enlarged to the left. A diastolic aortic murmur transmitted to the ensiform cartilage; a systolic murmur conducted to the axilla and the scapular angle. (Compare Figs. 172, 181.)

In addition to the rumbling presystolic murmur heard near the cardiac

in mitral obstruction, a soft, blowing, early diastolic murmur is
n heard along the left sternal margin. This murmur is due to dilata-
of the pulmonary artery which, if sufficiently marked, causes an in-
ciency of the pulmonary leaflets. (Graham-Steell murmur.)



IG. 195.—Diagram illustrating the direction of the regurgitant blood stream in *aortic iciency*. The murmur is best heard at the second right costo-sternal articulation and nsmitted toward the ensiform cartilage and toward the cardiac apex.



IG. 196.—AORTIC INSUFFICIENCY. The dotted areas indicate the locations over which iastolic murmur may be heard. The circle contains a diagrammatic representation e murmur in relation to the first and second heart sounds

Thus mitral stenosis complicated by relative pulmonary insufficiency
closely simulate aortic insufficiency with a Flint murmur. But in
latter case left ventricular hypertrophy is greater, the blood-pressure
ure is quite characteristic, and other pulsatory phenomena such as a
llary pulse, Duroziez's murmur, etc., will establish the diagnosis.

The difficulty so many students have in recognizing or even being
to identify presystolic murmurs is due to the fact that they expect

to hear a swishing sound similar to that produced by systolic or diastolic murmurs. The presystolic murmur is a sound very different in quality. It is a *rumble* somewhat like a short roll on the drum, which precedes and gradually merges into the first sound of the heart.

When the heart becomes dilated and the auricle paralyzed the murmur disappears, and fibrillation of the auricle makes its appearance. The crescendo quality while clinically characteristic has been shown to be due, not to an inherent quality of the murmur but chiefly to the proximity of the first heart sound. If the auricle goes into a state of fibrillation or if it loses its contractile force because of extreme dilatation, the presystolic murmur disappears and instead of it a murmur is heard early in diastole. *Pure presystolic murmurs can arise only at the auriculo-ventricular orifices.* Phonetic equivalent: R-R-R-upp, Dupp (presystolic rumble); Rup-Tut-Rarou (mid-diastolic rumble) (see Fig. 202).

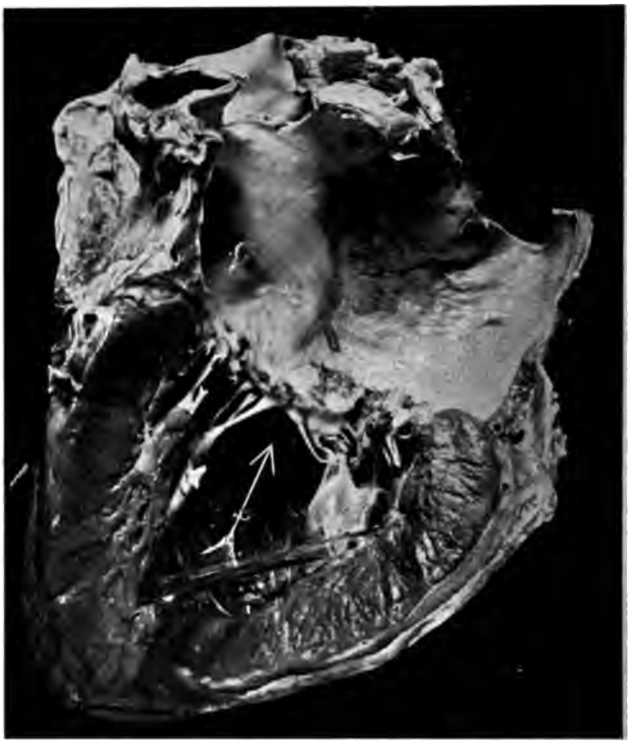


FIG. 197.—MITRAL INSUFFICIENCY. The mitral curtains are thickened, and are the seat of numerous vegetations. Some of the chordæ tendineæ are ruptured, all of them are thickened and shortened. The left ventricle is hypertrophied. The murmur of mitral insufficiency is systolic in time. It is transmitted toward the left axilla and scapula in reverse of the direction of the blood stream by the papillary muscles and the chordæ tendineæ. The arrow indicates the direction of the regurgitant blood stream.

5. The systolic tricuspid murmur is heard at the tricuspid area, because both the right auricle and ventricle lie immediately beneath this point. It is also often heard over the mitral area, and cannot always be

ferentiated from the murmur of a mitral insufficiency, with which it usually associated. The presence of marked right heart dilatation, a positive venous pulse, of an enlarged or even pulsating liver, pulmo-
ry congestion, ascites, etc., enable us to definitely state that the

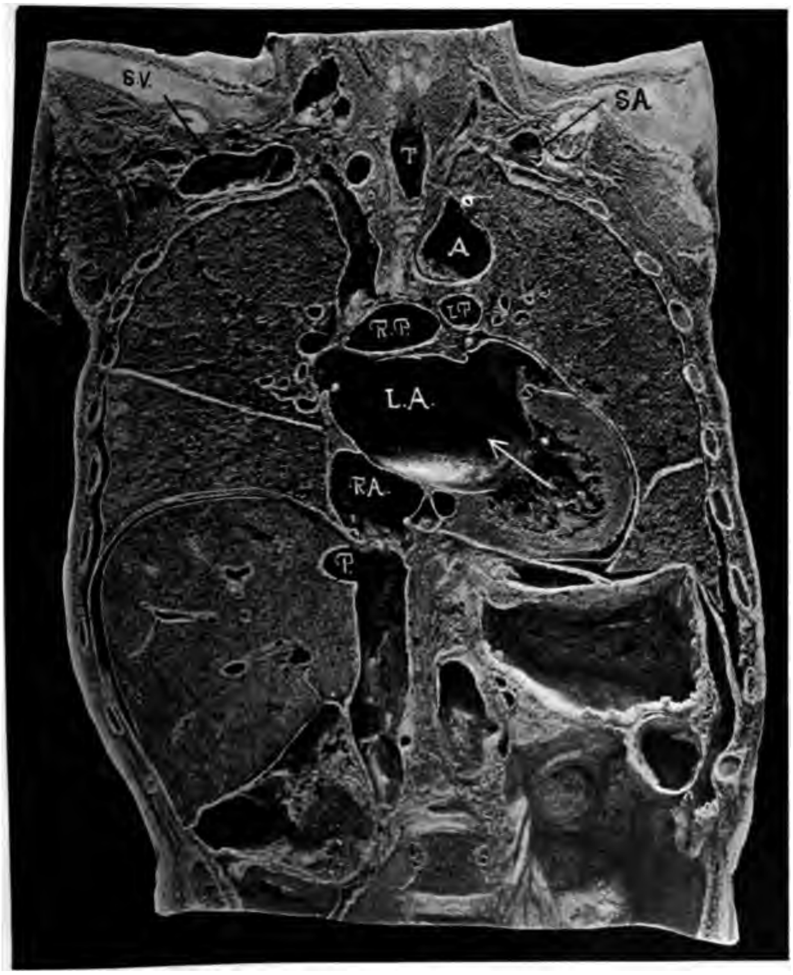


Fig. 198.—Mitral insufficiency. The left ventricle is hypertrophied, the left auricle ated. The direction of the regurgitant blood flow, as indicated by the arrow, is upward l backward, but the murmur is transmitted chiefly downward and forward through the rating chordæ tendineæ and the papillary muscles. *A*, aorta; *L.A.*, left auricle; *R.A.*, t of right auricle; *P*, portal vein; *R.P.*, right pulmonary artery; *L.P.*, left pulmonary ery; *T*, trachea; *S.A.*, right subclavian artery; *S.V.*, right subclavian vein.

cuspid valve is incompetent. A diagnosis of tricuspid insufficiency, sed merely upon the fact that a systolic murmur is clearly audible at e tricuspid area is not justified. Such a diagnosis may on the con-
ry be eminently warranted in the absence of a tricuspid murmur if

the presence of a positive venous pulse or a pulsating liver c demonstrated. Phonetic equivalent: Luf-f-f Dup (see Figs. 211, 4

6. **The presystolic tricuspid murmur** is heard at the tricuspid ar results from obstruction of that orifice (see Fig. 202). This, as an is lesion, is very rare. It is usually associated with mitral obstruction of "rheumatic" origin. A large "a" wave in the liver pulse is said suggestive of the condition. A few cases have been diagnosticated life; most of the cases found at autopsy go to the table with a dia of "mitral obstruction."

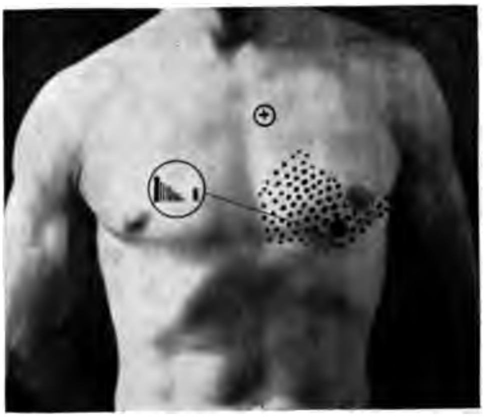


Fig. 199.

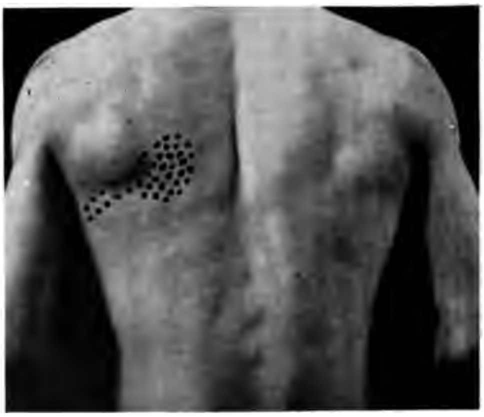


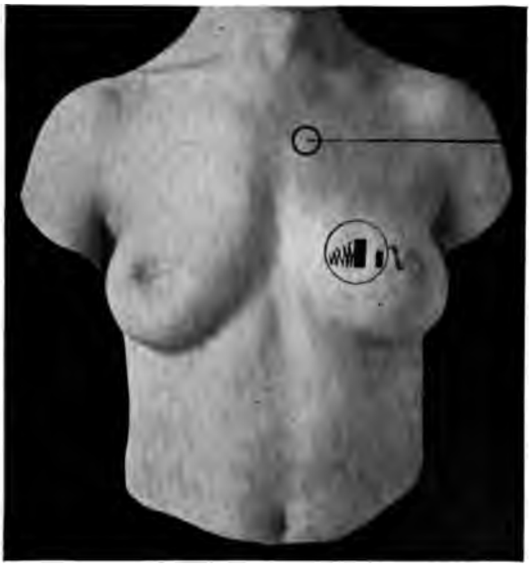
Fig. 200.

Figs. 199 and 200.—Mitral insufficiency. The dots indicate the area over w systolic mitral murmur is generally audible. The small circle indicates the acce pulmonic second sound (pulmonary arterial hypertension). The large circle co diagrammatic representation of the relation of the murmur to the heart sounds. W it is often audible at the angle of the left scapula.

7. **The systolic pulmonic murmur** if organic in nature is general to a congenital deformity of this orifice which results in obstructio

d and best heard at the pulmonic area and may be transmitted to ıbclavicular region (along the pulmonary arteries). It is associated marked cyanosis, clubbing of the fingers, dyspnea and if the child long enough, often with pulmonary tuberculosis. It is accompanied ıystolic thrill at the base of the heart, a weak pulmonic second sound, hypertrophy of the right heart. Phonetic equivalent: Luf-f-f Dup Figs. 1, 209, 329).

ſany functional murmurs arise at the pulmonary orifice, and *most ıonic murmurs are functional.* Experimentally, when the heart is ısed, the slightest compression or traction upon the artery is sufficient



ı. 201.—Mitral obstruction. Showing the area over which the diastolic (presys- nitral murmur is generally audible, together with a diagrammatic illustration of its n to the heart sounds. Accentuation, splitting or reduplication of the pulmonic sound is an invariable accompaniment of this lesion until compensation fails.

ıduce a murmur. Such pressure may easily be produced in health ange of posture, forced held inspiration, slight pleural adhesions, The pulmonary area was named by Balfour the "region of romance" se so many, variable, inconsequential murmurs may be heard in egion. There are, however, some cases of mitral insufficiency in the regurgitant murmur is heard near the second left intercostal owing to the proximity of the left auricular appendage (Figs. 70).

liagnosis of pulmonary stenosis based solely upon the presence of a ic murmur at the second left intercostal space is absolutely un- ed. *Pulmonary stenosis is a rare and nearly always, congenital* which is associated with cyanosis, clubbed fingers, cardiac hyper- r and a feeble second sound.

The diastolic pulmonic murmur (pulmonary insufficiency) may from congenital disease, or from ulcerative endocarditis; in either is a rare lesion. It may be due to functional dilatation of the pul-

monary orifice under severe strain which leads to increased blood-pr
in the lesser circulation. The *Graham-Steell murmur* is a dia
murmur heard best at the third left intercostal space. It is usuall
and blowing in character, replaces the pulmonic second sound, a
often heard only in recumbency. It may be influenced by respir
and by stethoscopic pressure (Goodman[1]) and may occupy part or
of the diastolic period. It is due to enlargement of the pulmonic rin
associated with dilatation of the right ventricle. It is of clinica

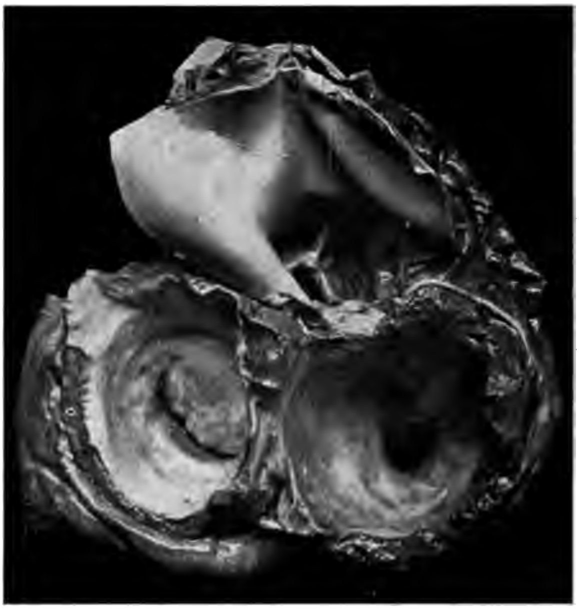


FIG. 202.—AORTIC, MITRAL AND TRICUSPID OBSTRUCTION. The lesions depicted a
end result of acute, generally rheumatic, endocarditis. The mitral orifice shows a typic
tonhole deformity with induration and calcification. The tricuspid orifice shows au
constriction, while the aortic leaflets are fused together and sclerotic, with seconda
careous infiltration. Tricuspid stenosis as an isolated lesion is rare. It may be due
same etiologic factors as the mitral lesion (generally rheumatic fever) or may be the
of the mitral lesion, which by overstraining the right ventricle leads to edema, hemo
and subsequent fibrosis of the tricuspid curtains.

The *physical signs* consist of a presystolic thrill and murmur, a right-sided cardi
largement, marked cyanosis and a presystolic liver pulsation. An intravitam di
is rarely made, owing to the similarity of the physical signs with those of mitral obstr
(*After Norris' "Studies in Cardiac Pathology."*)

portance inasmuch as aortic insufficiency with a Flint murmur may
sent identically the same acoustic phenomena as mitral stenosis w
Graham-Steell murmur. It is differentiated from the aortic regur
murmur by the fact that it is not transmitted to the ensiform car
nor toward the apex, and that it is unassociated with a Corrigan
and with the blood-pressure picture so characteristic of the latter l
Phonetic equivalent: Lub-Duf-f-f.

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[1] GOODMAN, E. H.: "The Graham-Steell Murmur in Mitral Stenosis." *Am
Med. Soc.*, Feb., 1919, 206.

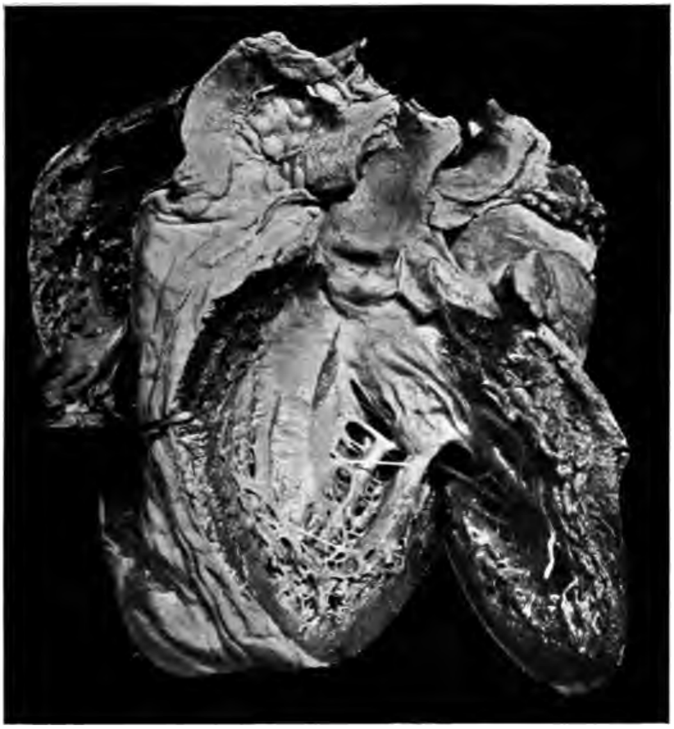


FIG. 203.—MITRAL STENOSIS. This lesion generally occurs in early adult life, and results rom rheumatic endocarditis. The illustration shows enormously thickened and contracted itral curtains, thickened, shortened and adherent chordæ tendineæ, and as a result, a nnel-shaped mitral stenosis.

The most characteristic *physical signs* of mitral obstruction are: a presystolic thrill and urmur at the apex, with a snappy first sound, and accentuation of the pulmonary second und. The pulse is small in volume and frequently irregular. The heart enlarges toward e right.

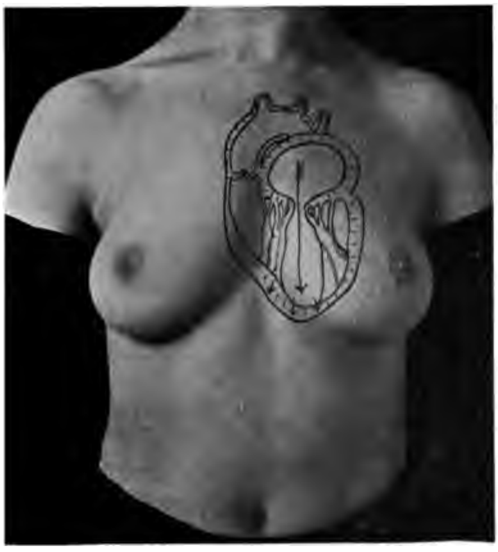


FIG. 204.—Illustrating *the direction of blood* flow in mitral obstruction.

All these rules are subject to exceptions due to cardiac displacement, etc. Murmurs themselves are variable and often multiple and complicated by pericardial or respiratory sounds. Occasionally murmurs are subjectively audible to the patient himself, or they may be heard by the examiner at quite a distance from the patient. Both of these statements apply chiefly in case of certain, loud, high-pitched, musical murmurs.

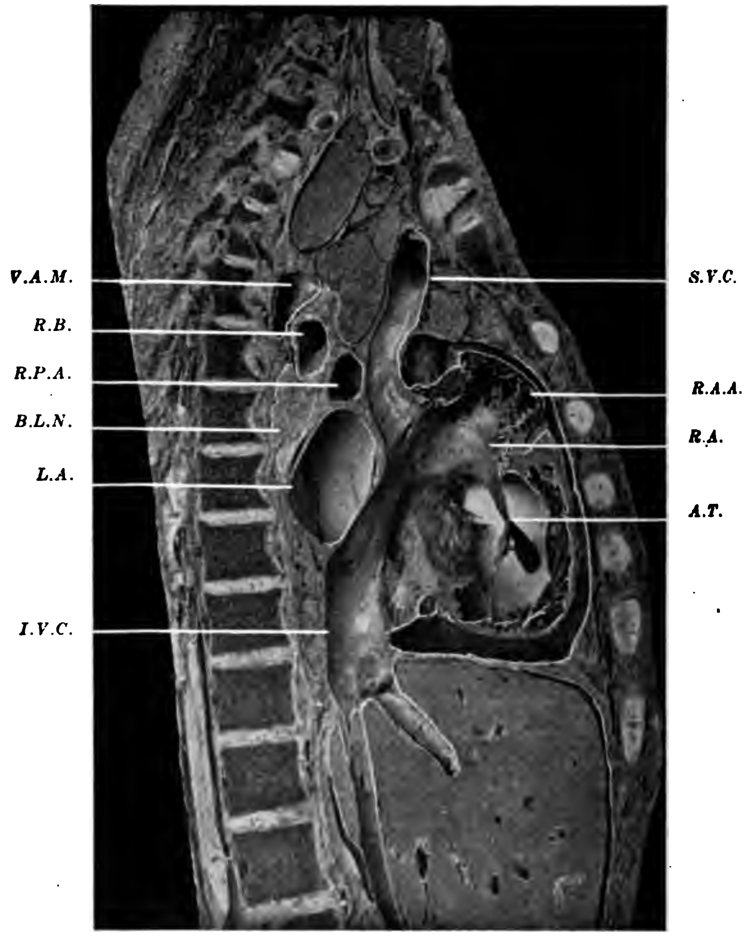
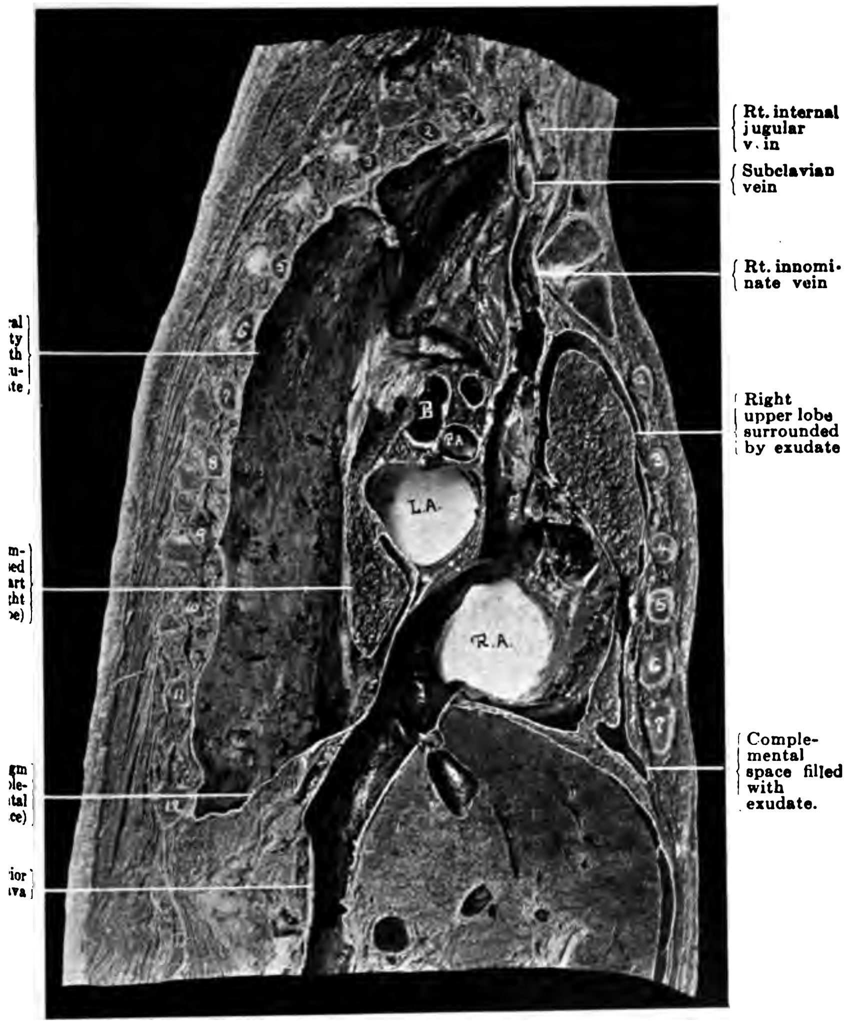


FIG. 205.—THE RIGHT HEART AND ITS TRIBUTARY BLOOD CHANNELS. *Pulsation is readily transmitted to the liver* either (1) directly from the right ventricle or (2) indirectly, as the result of tricuspid regurgitation, the inferior vena cava being short. In the latter instances the pulsation instead of being more or less localized to the epigastrium is general and expansile in character. The liver shows sphygmographically a positive venous pulse.

The blood pathway from the jugular veins into the right auricle is also a very direct one, hence jugular tracings (phlebograms) readily depict pressure changes in the auricle, especially when the subject is recumbent and gravity assists in the stasis of venous flow. *R.B.*, right bronchus; *R.P.A.*, right pulmonary artery; *B.L.N.*, bronchial lymph node; *L.A.*, left auricle; *I.V.C.*, inferior vena cava; *S.V.C.*, superior vena cava; *R.A.A.*, right auricular appendage; *R.A.*, right auricle; *A.T.*, anterior tricuspid leaflet; *V.A.M.*, vena azygos major.


Rt. internal jugular vein
Subclavian vein
Rt. innominate vein
Right upper lobe surrounded by exudate
Comple-mental space filled with exudate.
al ty th u- ite
m- ed art ht e)
gm le- ital ce)
ior va
B
P.A.
L.A.
R.A.

'IG. 206.—RIGHT-SIDED HYDROTHORAX. Frozen section from a case of cardiac anasarca ar to that depicted in Figs. 211, 381. Almost the entire pleural cavity was filled with us effusion. The lung which was completely collapsed and compressed, has been re- ed in the proximal sections, leaving only small areas in this, the last section cut. The t is pushed forward, the diaphragm and liver downward. The inferior vena cava is ly distended, the right auricle dilated. The liver is engorged and has the typical meg" appearance. The complemental space anteriorly and posteriorly was filled fluid. The patient, who suffered from rheumatic mitral obstruction and insufficiency, tricuspid insufficiency with auricular fibrillation, died with cyanosis, orthopnea and ral anasarca. *L.A.*, left auricle; *R.A.*, right auricle; *B*, bronchus; *P.A.*, pulmonary y.

FUNCTIONAL MURMURS

The term "functional" murmur is applied to various (almos versally systolic) sounds which cannot be explained by demonst structural lesions. The term is an unfortunate one because mu are physical phenomena and must result from physical causes. A may leak from mere lack of tonus of the surrounding muscular While this may be temporary, remediable and inconsequential, nev less it is a leak. It would seem better therefore to drop the term " tional" altogether and assign the murmur to its cause—endoca myocardial dilatation, anemia. Cardiac displacement and exophth goitre are often associated with systolic murmurs, as are also the va forms of anemia.

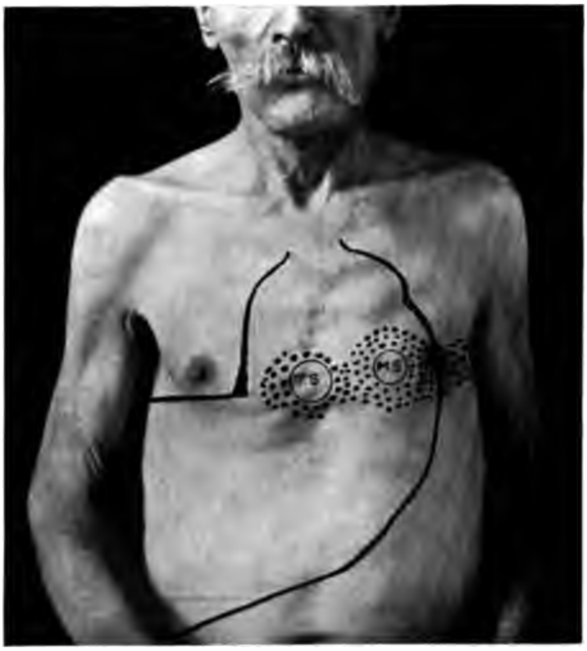


FIG. 207.—MITRAL AND TRICUSPID INSUFFICIENCY. The heart is enlarged, as is al liver, which is tender and which pulsates. The dotted areas show the regions over whi systolic mitral and tricuspid murmurs are heard.

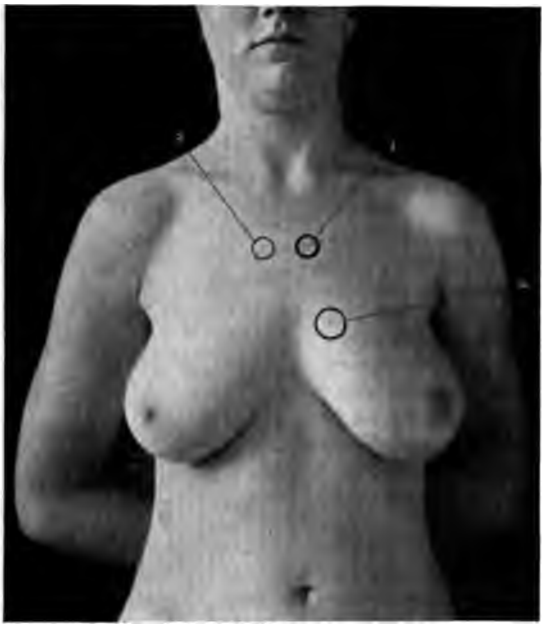
The cardiac murmurs heard in anemia have certain character which, as a rule, serve to distinguish them from murmurs due to ana ical changes in the heart.

1. They are definitely associated with the heart and while they be heard at any one of the valvular orifices, they are most frequ encountered over the pulmonary area, at the second costal cartila third interspace on the left. Next to the pulmonary area the regi the apex of the heart is the most frequent site. Occasionally a l murmur is heard over the aortic and tricuspid areas.
2. They are not transmitted but are sharply localized.

Γhey are always systolic in time, but often terminate before the systole.

They are frequently associated with a hemic murmur either veins or the arteries.

They are uniformly low-pitched; the sound is soft in character, riable in quality and intensity. The sound produced by a hemic ır never has the rough or occasional musical quality of an organic ır. At times these murmurs are loudest when the patient is in cumbent posture and in some cases this is the only position in it can be elicited. The murmur is apt to be loudest immediately lying down, before the vessels have accommodated themselves to the e in gravity. The murmur may be intensified by violent cardiac



ı. 208.—Locations at which non-organic murmurs are most frequently heard.

due either to nervous excitement or exertion. Hemic murmurs vessels of the neck are most marked in the erect posture.

ısitory Murmurs Due to Exertion.—Accidental murmurs aply endocardial in origin are frequently developed as the result of exertion. They are very transitory and are rarely heard beyond minutes succeeding the exertion. In an examination of 1552 ts at Cornell University, Munford encountered murmurs of this ter in 117, or 7.48 per cent., after the student had been required in" himself as often as possible.[1] They are generally ascribed to

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[1] T. McKenzie found that among 266 apparently healthy students 74 (27.8 per eveloped cardiac murmurs under vigorous exercise. Of these 35 were of the ımonic systolic type. If the cases are included in which this murmur occurred iation with other murmurs it was present in 64 cases, only 10 men out of the g to show it. Murmurs were slightly more common among the non-athletic

loss of muscle tone affecting chiefly the mitral sphincter and hence are more common in individuals who are not "in training."

Heart murmurs are very easily produced. The vascular dilatation induced by prolonged sweat baths is of itself sufficient to cause the appearance of murmurs in the majority of cases.[1]

THE EFFECT OF RESPIRATION ON ENDOCARDIAL MURMURS

1. *Inspiration* causes the lungs to overlap the heart and thus tends to make murmurs more feeble.

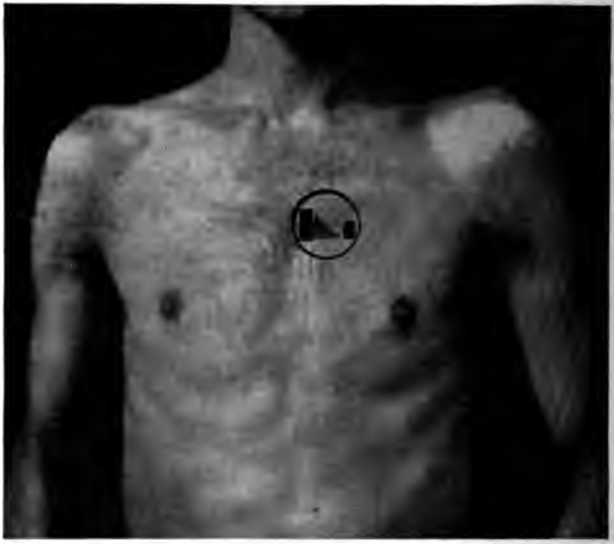

FIG. 209.—Pulmonary obstruction, or dilatation. Indicating the area over which systolic pulmonary murmurs are heard, with a diagrammatic illustration of the relationship between the murmur and the heart sounds.

2. *Inspiration* favors blood flow into and out of the right heart. During rapid breathing it hinders blood flow in the left heart, by retaining more in the lungs. During slow breathing this effect is only manifest during the first half of the act, because in the last half the pulmonary vessels dilate no further.

3. *Expiration* acts in the reverse manner, hindering blood flow in the right and favoring it in the left heart, especially with rapid or forced breathing. These facts may be used to help in the differentiation of right- or left-sided valvular lesions.

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students. These murmurs were more frequent, more intense and sometimes present only in recumbency.

[1] HOWELL, F.: "Physiological Heart Murmurs Produced by Electric Light Baths." *Boston Med. & Surg. Jour.*, April 3, 1902.

SPECIAL VARIETIES OF MURMURS

Duroziez's Murmur.—If the femoral artery be slightly indented by pressure of the stethoscope during auscultation, a systolic murmur will be heard which results from vibration of the vessel walls due to the local constriction of the blood stream. In aortic insufficiency, where blood flow is rapid and where the current flows alternately forward and backward, a *double murmur* may be heard *over the femoral artery.* This is known as Duroziez's murmur. It is suggestive but not pathognomonic of aortic insufficiency. It may occur in other conditions associated with vasomotor relaxation.

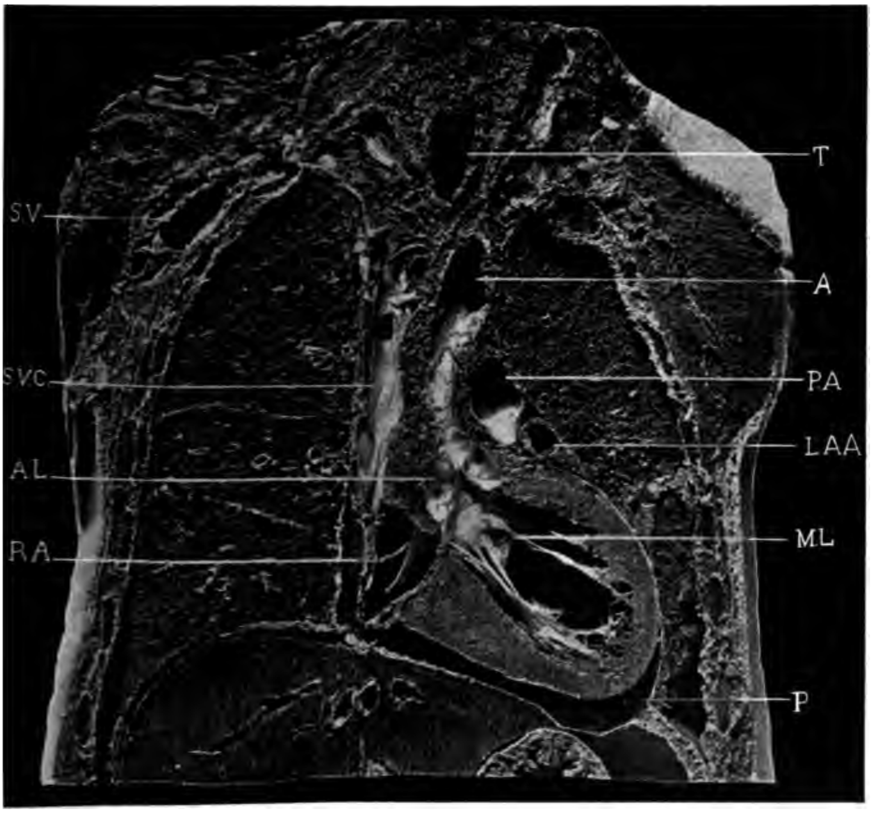

FIG. 210.—AORTIC AND MITRAL INSUFFICIENCY. Aortic and mitral insufficiency with left ventricular hypertrophy and dilatation, also a small pericardial effusion.

The aortic valves are shrivelled and retracted, the left ventricle is hypertrophied and dilated, this dilatation affecting the mitral orifice. The apex impulse is displaced downward and to the left. *T*, trachea; *A*, aorta; *P.A.*, pulmonary artery; *L.A.A.*, left auricular appendage; *M.L.*, mitral leaflets; *P*, pericardial sac; *S.V.*, subclavian vessels; *S.V.C.*, superior vena cava; *A.L.*, aortic leaflets; *R.A.*, right auricle.

Physical signs: A heaving impulse, a booming first sound, a systolic mitral murmur transmitted to the axilla. A faint, low-pitched aortic diastolic murmur transmitted toward the cardiac apex and to the ensiform cartilage. Pulmonic second sound accentuated. Pulsating carotid and brachial arteries, a water-hammer pulse, Duroziez's murmur, blood-pressure: systolic 180, diastolic 55 mm. Hg.

Traube's Sound.—In some cases of aortic insufficiency with marked hypertrophy and a large systolic output, the sudden distention of the arteries produces a cracking noise known as a pistol-shot sound. This may be heard even in the absence of aortic insufficiency if the pulse pressure be large, and the diastolic pressure low. In some cases of *aortic and tricuspid insufficiency* a *double sound* of this character (Traube's sound) may be heard over the femoral vessels. This has been shown to consist of a venous as well as an arterial element. It is due to sudden distention of the femoral artery, and to sudden backward pressure upon the femoral venous valves. The latter element precedes the former because the blood from the leaking tricuspid valve begins to regurgitate during the period of valvular closure (beginning of systole) while the arterial distention does not take place until the end of this period.[1]

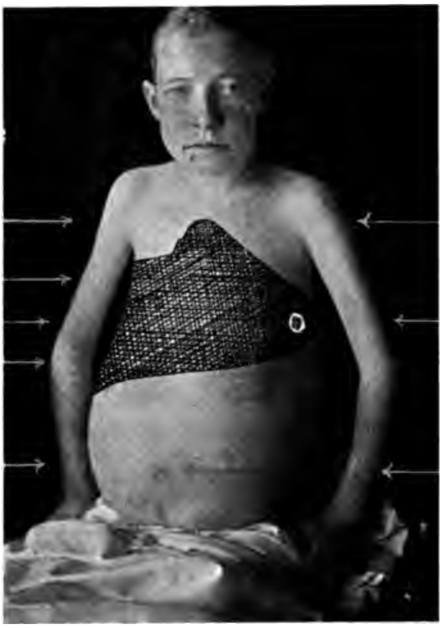
Skodaic tympany, exaggerated breath sounds, crackling râles.
Dulness, feeble breath sounds, fremitus and vocal resonance.
Flatness
Pulsating liver tenderness.
Bulging flanks, succussion wave.
Pulmonic second sound loud.
Apex impulse displaced, downward and outward arrhythmia.
Systolic murmur replaces first sound.
Pitting of the abdominal wall, edema of arms, glossy skin.

FIG. 211.—CARDIAC DROPSY. A case of general dropsy (anasarca) following dilatation of the heart, due to mitral and tricuspid disease. Orthopnea, cyanosis, right-sided hydrothorax, passive congestion of the lungs, liver and spleen, ascites, etc. The patient, a girl of fourteen years, shows emaciation and retarded development due to the cardiac lesion. Also pitting of tne abdominal wall due to subcutaneous edema. A characteristic picture of broken cardiac compensation with tricuspid insufficiency is presented (comp. Figs. 176, 205).

The Flint Murmur.—This murmur is *presystolic* in time; it is best heard within and above the cardiac apex. It occurs in some cases of *aortic insufficiency.* There has been much dispute about its etiology and significance, but it is generally believed to be due to the flapping of the anterior mitral curtain between two blood streams, *i.e.*, between the normal flow from the left auricle and the regurgitant flow from the ortic leaflets into the left ventricle. Hirschfelder has shown that in the xcised heart, if the ventricular pressure is increased, the mitral valve pens only along part of its line of closure, and that therefore an actual unctional stenosis may exist. The importance of this murmur lies in he fact that *it may present the exact acoustic phenomena of organic nitral stenosis.* It may be differentiated from the last-named condition y the fact that it is not associated with a marked presystolic thrill, an nlarged right heart, a snappy first sound and a small, well-sustained ulse. The Flint murmur is found in conjunction with other signs of ortic insufficiency such as: a pulse which is large in volume and collapsing in type—the diastolic pressure being low and the pulse pressure arge; left ventricular hypertrophy and the vascular and pulsatory henomena so characteristic of an incompetent aortic valve[1] (see Fig. 212).

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[1] SCHULTZE: *Deut. Med. Woch.*, 1905, xxxv.

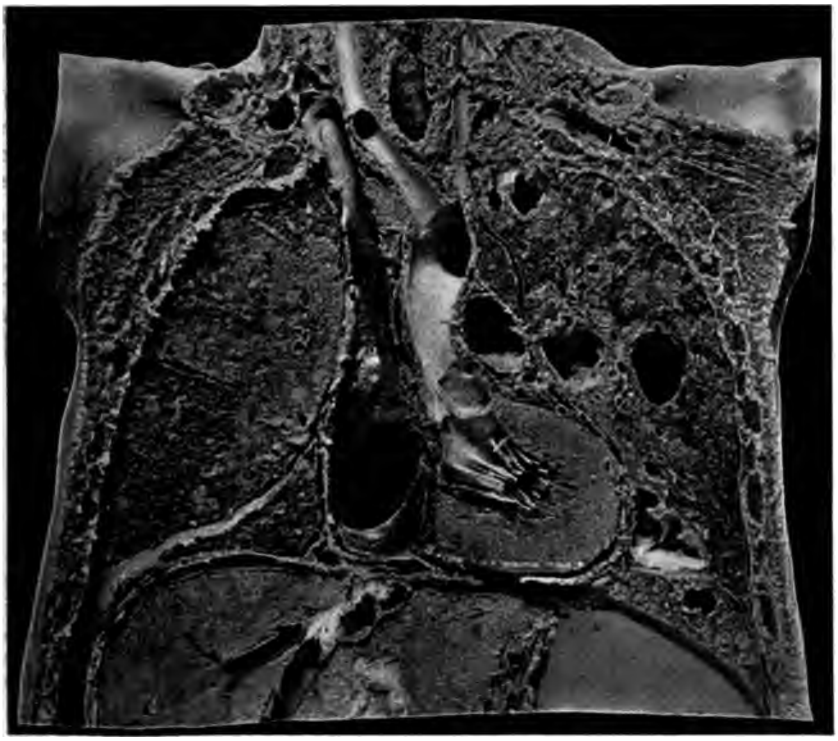

Fig. 212.—The Flint murmur. The aortic and the mitral valves are contiguous tructures. The Flint (presystolic) murmur is produced by the regurgitation of blood hrough the aortic orifice. The curtains of the mitral valve are caught between two blood urrents—that just mentioned and that streaming in from the left auricle—and flap like a oose sail in the wind.

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[1] The relative frequency of *pulsatory phenomena* in 124 cases of aortic insufficiency was found to be as follows: Corrigan pulse 95 per cent., capillary pulse 90 per cent., Duroziez murmur 88 per cent., visible arterial pulsation 76 per cent., femoral snap 5 per cent., Traube's sign 24 per cent., visible venous pulse 7 per cent. (Tice).

Roger's murmur is sometimes heard in cases of *patulous interventricular septum.* It is continuous throughout systole and diastol with an ictus or reinforcement during the former. It has a rhythmic somewhat musical character with a periodic intensification like th whirring of a foot-propelled knife-grinder's wheel. It is best heard i the upper third of the precordium over the median portion of the septu itself. It is not transmitted but gradually fades out in all directions a the point of maximum intensity is receded from. Phonetic equivalent Whir -r -r -r -r (Fig. 88, I).

EXOCARDIAL MURMURS

In the large arteries near the heart (subclavian, etc.) certain sound may be faintly heard. They may be due to: (1) stretching and vibra tion of the vessel walls, or to (2) conduction of the second heart soun In the distant arteries one may have murmurs from stethoscopic pre sure, or murmurs conducted from the aortic orifice, etc. Very often systolic murmur is heard over the vertex of the skull of children fro the third month to the sixth year. It is without significance.

Systolic murmurs in arteries may be due to: (*a*) roughening of th intima; (*b*) narrowing of lumen; (*c*) dilatation—fevers, vasomotor pa esis; (*d*) aneurism.

Subclavian murmurs are generally short in duration and alwa systolic in time. Often a harsh systolic whiff is heard, especially durin inspiration, generally on the left side, and most commonly in men the junction of the middle with the outer third of the clavicle. Th murmur is increased by deep inspiration, sometimes audible only durin forced inspiration and modified by the position of the arm. It ma be inconstant. Subclavian murmurs possess *no pathologic significan* they are generally due to constriction of the artery between the clavic and the first rib, although a similar constriction may be produced b fibroid disease of the pleura. Hence they are frequently encountered i cases of pulmonary tuberculosis. Landis found, among 31 cases studie that the murmur was associated with pulmonary tuberculosis in 2 The fact that the murmur is heard more frequently on the left than t right side, even in cases in which the pulmonary disease is more extensi on the right side, suggests that the real explanation lies in some anatom cal variation of the two arteries. Furthermore, subclavian murmu have been frequently noted in individuals healthy in every respec While probably of but little importance the presence of a subclavia murmur should suggest a careful examination of the apices of the lungs.

Carotid Murmurs.—Systolic murmurs heard in the carotid arterie especially the right, are not uncommon. As a rule the murmur is tran mitted, having its origin in disease of the aortic leaflets (stenosis o roughening) or in disease of the first part of the aorta (roughening fro atheromatous plates or slight dilatation). The latter conditions are b far the most frequent sources of the murmur. Occasionally a systoli murmur, due to anemia, is heard in the carotids. An aneurism ma produce a similar murmur.

Murmurs are frequently heard over the enlarged thyroid gland i *exophthalmic goitre.* They may be systolic or diastolic in time.

ARTERIAL SOUNDS AND MURMURS

Sounds and murmurs are frequently audible over the larger arteries of the body, both in health and in disease. Regarding the etiology of the "sounds" there is a difference of opinion. According to one view these sounds originate in the heart; the first sound resulting from mitral, the second from aortic closure, and are conducted by the blood stream and the vascular wall. The opponents of this hypothesis maintain that the first sound arises locally, as a result of distention and subsequent vibration of the vascular wall; and that the second sound is transmitted from the aortic valve. Sounds originating at the aortic orifice are usually audible in the carotid, subclavian and sometimes brachial and femoral arteries. The height of blood pressure seems to have but little effect upon conduction unless the pulse pressure is large, in which case the likelihood of local arterial sound production is much enhanced. It is of course important to determine whether the sound heard occurs *only* over the artery or generally over the surrounding tissues as well.

There seems to be no great difference in regard to whether the arteries are normal or sclerotic, although experimentally glass and elastic metals conduct sounds further than rubber, but this depends to a considerable extent upon the character of the surrounding media. If the tissues surrounding a tube are physically more or less similar to the tube the sound will be diffused in all directions, and far less of it will reach a distant point than if the tube is surrounded by a medium of different density, such as air. The statement is often made that sounds are conducted better in the direction of the blood stream. This factor is of but little importance. Sounds are conducted by the vascular wall and by the column of blood, the *direction* of the blood flow is unimportant.

Murmurs heard in the arteries may be conducted from the heart, but before assuming this one must be sure that they have not been artificially produced by local pressure of the stethoscope upon the arterial wall. The murmur of aortic obstruction is often well and widely heard in the large aiteries but it is sometimes extensively heard over other tissues as well. Occasionally heart sounds may be advantageously studied in the carotid artery when they are almost inaudible in cases of emphysema or over-shadowed by pericardial friction sounds (Roberts[1]). Local abnormalities, aneurisms, especially the arterioveneus variety, or neighboring tumors causing arterial constriction, of course also give rise to arterial murmurs. Nearby veins may give rise to bruits which may be mistakenly attributed to the arteries.

VENOUS MURMURS

Venous murmurs (bruit de diable, nun's murmur, souffle, venous hum) were formerly considered an indication of anemia. They consist of soft, low-pitched continuous humming sounds of variable quality and intensity with a rhythmic accentuation. They are heard in the large veins above the clavicles in 50 per cent. of all children. They are loudest during auricular diastole (faster flow), in the erect or sitting postures, and on the right side behind the lower margin of the sternomastoid muscles.

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[1] Roberts, S. R.: "A Study of Arterial Sounds." *J. A. M. A.*, lxix, Sept. 15, 1917, 873.

The right internal jugular vein is almost a linear continuation of the right innominate vein, the internal jugular is held open by the cervical fascia and hence cannot diminish in calibre when the amount of blood lessens; hence a fluid vein is produced and a murmur results. The condition is increased by hydremia (anemia) (Figs. 136, 212).

Another explanation is that based on a constriction of the vein. "By having the patient turn the head upward and away from the side auscultated the internal jugulars are made tense and compressed against the transverse processes of the lower cervical vertebræ with which they are in relation. In this way the calibre of the vein is narrowed and the flow of blood accelerated."[1] Many explanations as to the exact cause of venous hums have from time to time been offered. None are invariably satisfactory. *The diagnostic value of venous murmurs from any standpoint is very slight.*

It is said that a venous hum may be heard in the femoral veins under the same conditions in which it is heard in the jugulars, but we have never been able to verify this statement.

Eustace Smith's Sign.—Occasionally a venous hum is heard over the sternum just beneath the notch. The murmur is elicited by tilting the head back and is supposedly produced in the left innominate vein, which passes from left to right with a slight obliquity downward behind the upper part of the sternum, by reason of the vein being pressed upward by enlarged bronchial lymph nodes. This explanation appears to be erroneous. The bronchial lymph nodes at the bifurcation of the trachea are too far above the heart to cause pressure on the large vessels. The pressure is probably caused by an enlarged *persistent thymus gland* (Gittings). When the murmur is present over the sternum it is also heard in the jugulars, where in all probability it originates.

Although frequently referred to as a sign of some value in the recognition of enlarged bronchial lymph nodes in children, we have rarely been able to elicit it even in cases in which there were other signs pointing strongly to this condition.

CARDIO-RESPIRATORY MURMURS

(Cardio-pulmonary, pseudo-cardial, cardio-pneumatic, systolic vesicular murmurs.)

Cardio-respiratory murmurs are *interrupted or abnormal breath sounds* produced by the movement of the heart upon the surrounding lung tissue. The respiratory sounds may be interrupted by a series of periodic interruptions or increments of intensity (puffs or "cogs") which may be heard even when the breath is held. They often vary with the cardiac or respiratory rate and phase. They are generally loudest near the cardiac apex and along the left border of the heart, but they may be heard along the right border, in the scapular region and elsewhere. They are influenced by respiration, being generally louder during deep inspiration. They are apt to be affected by change of posture; they generally disappear in recumbency, and nearly always during apnea. They are not accurately synchronous with the heart, but often appear in the middle of systole, being distinctly separated from the first sound. They often in and end suddenly, are sharply localized and seem close to the ear he examiner. They are not transmitted in the directions which are racteristic of endocardial murmurs. They are often intermittent, ociated with râles, and influenced by stethoscopic pressure. They y occur in perfectly normal individuals, but are especially common in monary tuberculosis, especially when the pleura has been extensively olved, and in atelectatic lungs, as well as in patients who have had uritis or pneumonia, or in those suffering from paroxysmal tachy-dia.

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[1] Landis and Kaufman: *Arch. Pediatrics*, Feb. 12, 1912.

Gerhartz has shown that cardiac murmurs are generally due to rations of about the same rate as those which correspond to the lower ge of the respiratory sound (60 to 80 vibrations per second). The act that the two types of sound have about the same pitch accounts for he difficulty which sometimes attends their differentiation. Cardio-espiratory murmurs have *no pathologic significance except that they may ndicate pleural adhesions; their importance merely lies in the fact that hey may be mistaken for endocardial murmurs or pericardial frictions.*

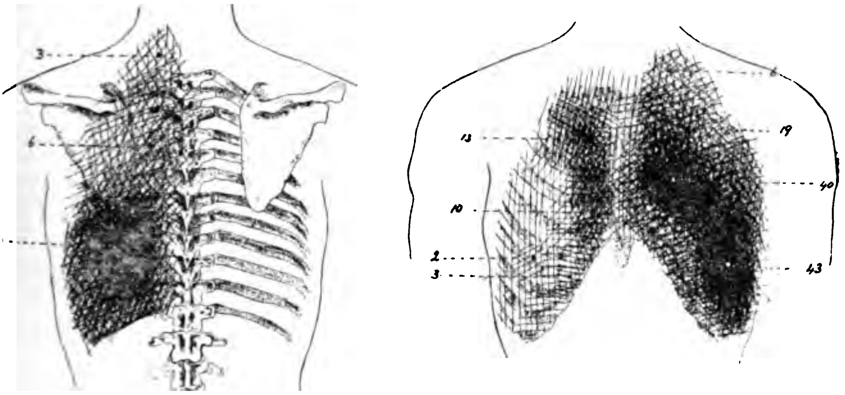


Fig. 213.—The distribution of cardio-respiratory murmurs in 48 cases. Composite ictures "constructed by shading the affected area of each chest upon the same diagram y lines approximately equidistant." The actual numerical frequency is shown by the ttached leaders. (*After Thomas Lewis, Quart. Jour. Med.*, Jan., 1909.)

Succussion splash synchronous with the heart may be heard in the eighborhood of the precordium. It is generally produced in the stom-ch, although it may be due to hydropneumothorax, or to hydropneumo-ericardium.

Hepatic murmurs may be heard over the liver or in the epigastrium n some (rare) cases of hepatic cirrhosis. They are generally venous in rigin (blood flowing from small into larger veins, or sinuses) and may be ontinuous or intermittent in character.

PERICARDIAL FRICTION SOUNDS

In the early stages of pericarditis the serous membrane which normally smooth and moist becomes roughened as a result of con-estion and exudation. The exudate, instead of noiselessly gliding over he neighboring pericardium, now produces a rough, scratching or scraping sound, similar in quality to that heard over an inflamed pleura. This sound is known as a pericardial friction. It *often has a dry, creaking or shuffling, leathery quality, is heard during both systole and diastole* and is therefore described as being "to and fro." It is frequently accompanied by local pain, seems close to the ear and is intensified by pressure. It remains localized, is not transmitted in the directions characteristic of endocardial murmurs, and is often loudest over the middle of the precordium where the heart is uncovered by lung tissue. It may be *differentiated* (1) from a *pleural friction* by the fact that it is synchronous

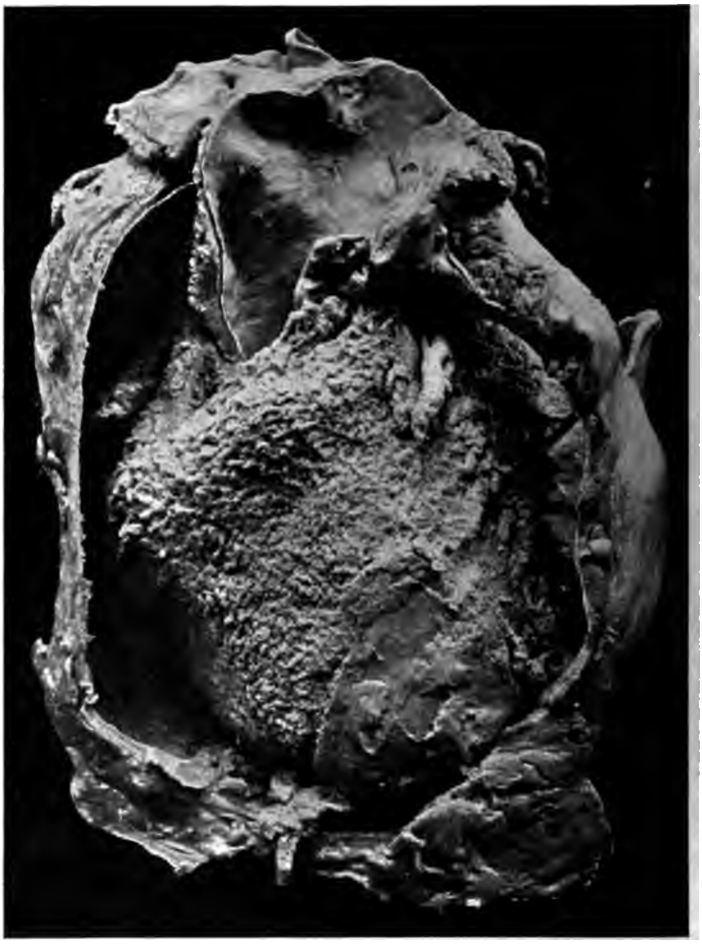
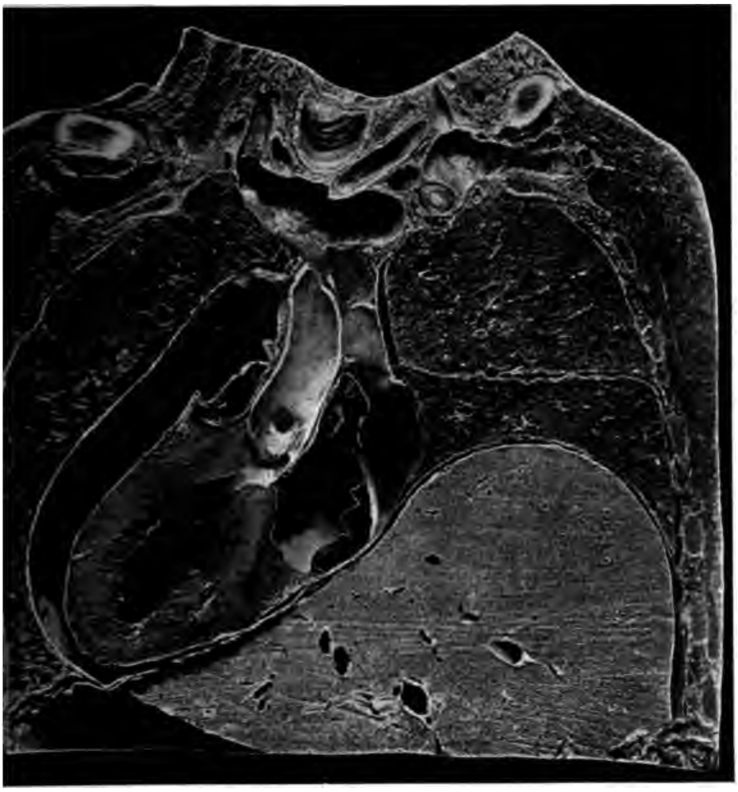


FIG. 214.—ACUTE SERO-FIBRINOUS PERICARDITIS. The pericardial sac is stretched and has been greatly distended by serous exudati. Both the mural and the visceral surfaces are covered with fibrinous exudate (cor villosum, bread and butter pericardium). Such a heart would yield loud friction sounds in the early stages of inflammation. Later when the serous surfaces were separated by liquid effusion the friction would disappear, except perhaps at the base, while the heart sounds would be muffled and distant. (*From Norris' 'Cardiac Pathology.''*)

eart, and not with respiration and that it does not disappear
Valsalva experiment; and (2) from *endocardial sounds,* by the
is not accurately synchronous with either systole or diastole,
verlaps them both because the greatest systolic excursion of
surface corresponds to the expulsion time and not to either
liastole. A further difference lies in the fact that the friction
ry in quality and intensity not only in the course of a few



.—**Pericardial effusion**: the body being frozen in the recumbent posture and
. *behind*. The heart is hypertrophied and dilated. Both lower pulmonar
mpressed, the left by the effusion, the right by the congested liver which has
upward as the result of ascites (recumbent posture). The physical findings
ı Fig. 216. (Compare Figs. 175, 377, 378.)

t even at times from one heart beat to another. Leaning for-
ying on the left side tends to intensify the sound while the right
cubitus often diminishes it. The pericardial friction may have
g triple rhythm very similar to gallop rhythm.

rdial "Knock."—During the recent war much interest was
y the "pericardial knock,"[1] a clicking sound heard over the
m in certain cases of penetrating chest wounds, in the neigh-
f the pericardium. This sound is sometimes audible not only

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[1] S. M Smith: *Br. Med. Jour.*, Jan. 19, 1918.

by the patient himself, but also by the examiner at some distai the patient. A clear cut metallic click, usually double in rhy synchronous with the heart; often transient and influenced by tion, it is best heard near the cardiac apex. The genesis of th startling sound has been ascribed to (*a*) emphysema of the me connective tissue or (*b*) free air in the interstitial connective tissu lung.

Pericardial Effusion.—Unless the pericardial inflammation is p arrested, the exudate poured out into the pericardial sac incre becomes more liquid in character—serum, pus or blood. Sucl cardial effusion muffles the heart sounds so that they become l even inaudible. Under such circumstances the friction sound dis

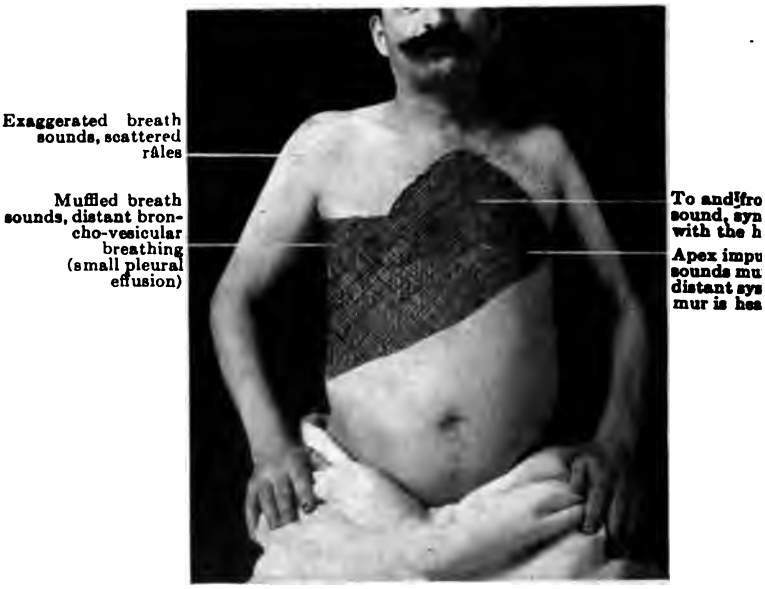
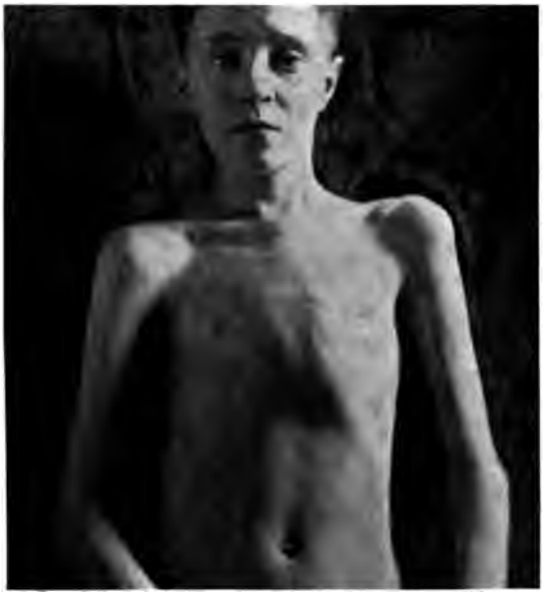

Exaggerated breath sounds, scattered râles
Muffled breath sounds, distant broncho-vesicular breathing (small pleural effusion)
To and fro sound, syn with the h
Apex impu sounds mu distant sys mur is hea

FIG. 216.—Percussion outlines in case whose heart is depicted in Fig. 215. dulness is very large and merges with that of the liver; it is somewhat pyriforn The cardiohepatic angle is obtuse. The abdomen is distended and bulges in (ascites). The patient suffers from orthopnea—the shoulders are raised and th muscles of respiration brought into play. The breath sounds were harsh and e and associated with crackling râles (congestion). The scar between the uml pubis results from paracentesis abdominalis.

Such a condition is associated with an increase of heart dulnes forms an obtuse cardio-hepatic angle. The inflammation and cause a softening and distention of the pericardium, which in so may become so large as to simulate a pleural effusion and ma compression of the left lung with associated dulness and bronchia ing. In children, bulging of the precordium may occur. Car ness extends further to the left beyond the apex impulse than is in hypertrophied hearts. Dulness in the fifth right intercos near the sternum (*Rotch's sign*) is sometimes demonstrable qu in the effusion stage. Stress is sometimes laid upon the fact dulness in pericardial effusions tends to be pyriform, but this is nly not always the case. An area of percussion dulness at the of the left scapula together with bronchial breathing, etc., fre- ;ly results from pressure of a large pericardial effusion upon a ɔn of the lung (*Ewart's sign*).

hanging the patient's posture from the erect to the right lateral ion tends, in pericardial effusion, to produce a greater displacement e heart to the right than is the case in simple hypertrophy.

rogressive enfeeblement of the heart sounds, especially when a on sound has been heard or suspected, may have distinct diagnostic e.



217.—Precordial bulging as a result of (rheumatic) pericardial effusion in a lad of 13 years.

t should be remembered that small pericardial effusions are fre- ıtly overlooked and it has been stated that no effusion is demon- ɔle by physical signs until it amounts to 150 c.c. One should be tantly on the lookout for evidences of pericarditis when dealing rheumatic fever and lobar pneumonia (see Figs. 377–382 incl.).

n X-ray examination may throw much useful light upon doubtful l.

'here is considerable variation regarding the *position of the heart in ardial effusions*, as well as difference of opinion regarding its cause. ems that the position depends upon the size of the heart, which in depends upon the amount of blood it contains, which is in turn de- ined by the stage of compensation. When the heart is well filled blood its position is relatively normal, but when it is small and partly psed it may fall backward against the posterior pericardial wall; the being more or less displaced toward the right. Occasionally it may remain in an anterior position, in which it is maintained by the elasticity of the great vessels.

Effusions amounting to 750 c.c. may be sufficient to cause the disappearance of all friction sounds even at the base, but on the other hand a friction may persist with effusions of 1000 c.c. or more.

Pleuro-pericardial sounds are due to roughening of the external surfaces of the pleura and the pericardium. They are heard best at the left anterior pulmonary border, are often affected by respiration, posture, etc.

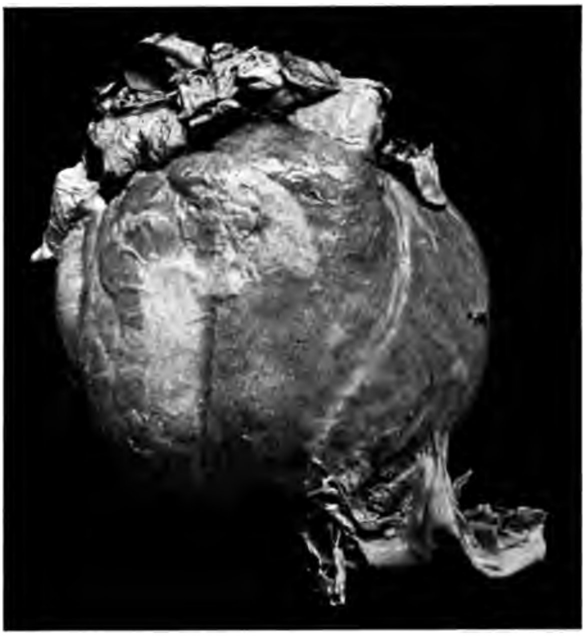


FIG. 218.—PERICARDIAL ADHESIONS. The illustration shows dense, localized, fibrous adhesions near the apex of the heart (which is dilated), the result of antecedent pericardial inflammation. Such a condition is suggested by localized systolic retraction of the chest wall, especially if associated with diminished postural mobility of the heart, with symptoms of cardiac insufficiency out of proportion to the demonstrable physical signs, lack of response to the usual methods of treatment (digitalis, rest, etc.), especially if coupled with a history of a previous attack of rheumatic fever or pneumonia. (*From Norris' "Cardiac Pathology."*)

PRACTICAL CONSIDERATIONS

In examining the heart, observations should always be made in both the erect and in the recumbent posture. The results thus obtained are often variable, and physical signs absent in one position may be readily demonstrable in the other. When the findings are recorded, the position should be stated.

In *the erect posture* the apex beat is lower, the cardiac dulness is lessened, the aortic second sound is louder than the pulmonic (after twenty-five years of age), the splitting of the second sound if previously present tends to disappear; the murmurs of aortic insufficiency and mitral obstruction become louder, as does also the venous vascular hum.

In *recumbency* the apex beat is higher, the cardiac dulness is generally more marked and the pulmonic second sound louder than the aortic, even in adults. Reduplication of the second sound is often present; the murmurs of mitral insufficiency, tricuspid insufficiency, aortic obstruction, and most functional murmurs become more intense. The differences are chiefly due to the effects of gravity upon the position of the heart and upon the blood flow.

It is often advisable to have the patient indulge in some form of *exercise*, provided his physical condition permits of it, such as climbing a flight of stairs, or "dipping" (touching the floor with the finger tips, by flexion of the knees and thighs). These procedures will often develop latent murmurs, irregularities or cardiac erythism. It is always well, especially in high-strung individuals, to count the *pulse* while the patient is resting quietly, before the clothes have been removed, and to repeat both the pulse count and the blood-pressure estimation at the end of the examination, after the patient has become tranquilized.

It should be borne in mind that *functional murmurs* are of frequent occurrence, and that *organic heart murmurs* are nearly always accompanied by demonstrable cardiac enlargement, increased pulse rate or other abnormalities. Further that "a disease of the valves is not a disease of the heart"; in other words as long as the myocardium is healthy the prognosis is relatively good regardless of valvular leakage or obstruction.

Regarding *blood-pressure* estimations we should bear in mind that the initial reading is often fallaciously high, especially in patients unused to this examination. Subsequent examinations made after the patient has learned the harmless and painless nature of the procedure are much more accurate. In these "psychic" elevations, however, the diastolic pressure is practically unaffected, even when the systolic pressure has been temporarily elevated 15 or 20 mm. Hg. Systolic pressures above 160 mm. and diastolic readings above 100 mm. Hg are abnormal at any age. The most frequent cause of persistent arterial hypertension is chronic glomerulo-nephritis. An examination of the urine is always essential since the cardiorenal relationship is a close and important one.

An examination of the *thyroid gland* will often explain cardiac abnormalities such as tachycardia, hypertrophy, dyspnea and lability of the pulse and of blood-pressure. Slight *edema* of the extremities at the end of the day point to cardiac fatigue and insufficiency. The possible presence of an *aortic aneurism* should always be borne in mind, in patients who complain of precordial pain, oppression, cough, dyspnea or other cardiac or respiratory symptoms without evident cause.

Upon discovering the existence of a murmur the examiner should determine (1) the area over which it is loudest; (2) its time in relation to the cardiac cycle; (3) the direction in which it is transmitted; (4) its character and constancy in relation to respiration, posture and exercise. It must be remembered that many entirely normal persons may present murmurs.

The effect of deep *forced inspiration and expiration*, each act being held for a number of seconds, should be noted. Functional and cardiorespiratory murmurs will be much affected by these procedures and may indeed disappear entirely during one or the other phase. Organic murmurs are much less or not at all affected. Furthermore, individuals with organic lesions generally cannot hold their breath as long as normal people without discomfort, distention of the jugular veins or cyanosis. The effect of *change of posture* from the erect to the recumbent position, and of *exercise* such as dipping, should always be noted not only in regard to the murmur but also upon the pulse rate, respiration and blood-pressure. The less marked and the more brief the changes produced, the less serious generally, the lesion. If the pulse rate remains high and the blood-pressure falls after moderate exercise, the individual is at least not in good training, and people with organic lesions naturally show the effect of a lack of it more than those with normal hearts.

Functional heart murmurs are never diastolic in time, they are not accompanied by other abnormalities such as hypertrophy, accentuation of the second sounds, arrhythmia, cyanosis, edema, etc., and they generally occur at the pulmonary area—the "region of romance" of Balfour.

Functional murmurs are not conducted in the same direction nor to the degree as organic murmurs. Thus a mitral systolic murmur if conducted to the angle of the scapula may be considered not only organic but the result of a well-marked lesion. A forcible cardiac impulse associated with a weak radial pulse bespeaks structural disease. The intensity of a murmur is not an index of the severity of the lesion, often quite to the contrary. Thus in *failing compensation* the murmurs become fainter and may entirely disappear, while the area of cardiac dulness increases; the apex beat becomes weaker and more diffuse, although sometimes more readily visible. Epigastric pulsation as well as that over the upper precordium becomes more intense, and accentuation of the pulmonic second sound disappears. It is frequently impossible to make an accurate diagnosis of the valvular lesions during broken compensation.

The *history* of an antecedent attack of rheumatic fever, chorea or tonsillitis is evidence in favor of an organic lesion, especially in case of the mitral valve; while the history of syphilitic infection or the presence of a positive Wassermann reaction more or less definitely settles the origin of a diastolic aortic murmur. The presence of marked anemia should make one very chary of declaring a murmur to be due to valvular disease.

It is to be remembered that *aortic and pulmonary stenosis* are rare lesions, whereas systolic murmurs over these areas are very common. At the aortic area systolic murmurs are usually due to a mere roughening or stiffening of the valves or to a slight aortic dilatation. At the pulmonary area systolic murmurs may result from the most trivial causes, such as pressure of the lung, traction of pleural adhesions, dilatation of the artery. Pulmonary stenosis is practically always a congenital lesion, associated with marked cyanosis, clubbed fingers, right-sided hypertrophy, congestion of the lungs, a weak second sound and thrills. Without some of these findings a diagnosis of pulmonary stenosis should never be made.

A diagnosis of *tricuspid insufficiency* cannot be made on the presence of a murmur alone. A positive venous pulse, pulmonary congestion or hepatic enlargement, edema, ascites, a pulsating liver, cough, dyspnea, if not orthopnea—at least some of these symptoms must exist before the diagnosis of tricuspid insufficiency is justified.

The presystolic murmur of *mitral stenosis* is generally characteristic and readily recognized owing to its rumbling quality and to the mitral ılse. It can be confused only with a Flint murmur or with the murmur tricuspid stenosis. The former is usually easily identified by means the blood-pressure picture, the marked left ventricular hypertrophy nd the pulsatory phenomena. Much more difficult is the diagnosis etween early mitral obstruction and a normal heart, with a loud preystolic element of the first sound, especially in cases of the Efful synrome, see p. 221. In some cases, even if the history, the symptoms, nd the physical signs are most carefully considered, experienced physiians will differ in their diagnosis and only time will decide the problem.

Tricuspid stenosis is a rare lesion, which when encountered is generally ssociated with mitral stenosis. But few cases have been correctly iagnosticated during life. For obvious reasons they are generally signed out" as mitral stenosis. The presence of an "a" wave in the ver pulse is said to be characteristic of tricuspid stenosis.

PART III

DISEASES OF THE BRONCHI, LUNGS, PLEURA, AND DIAPHRAGM

BY H. R. M. LANDIS, A. B., M. D.

CHAPTER XXI

DISEASES OF THE BRONCHI

ACUTE BRONCHITIS

An acute catarrhal inflammation of the mucous membrane of the trachea, large and medium-sized bronchi. In healthy adults it is, as a rule, a harmless affection, but in the very young and very old it is often a serious matter. In the latter age periods the inflammation shows a decided tendency to extend downward into the fine bronchi, leading eventually to a capillary bronchitis, or, as it is preferably called, a broncho-pneumonia.

Etiology.—As met with under ordinary conditions, acute bronchitis is of itself of relatively little importance, and except under the conditions above mentioned, it is rarely serious. It should be kept in mind, however that it is very commonly associated with the acute infections, particularly, typhoid fever and malaria, even from the beginning of these fevers. Acute bronchitis is so universally associated with measles that it should be considered a symptom rather than a complication. During the course of an epidemic of measles or influenza the bronchial inflammation may be very severe. In such instances the condition is characterized by a profuse, purulent bronchorrhea and the constitutional evidences of a severe toxemia. Secondly broncho-pneumonia, lobar pneumonia or pleural involvement often occurs. The presence of adenoids and enlarged tonsils are important predisposing factors in children (Holt).

It is of the greatest importance to keep in mind that tuberculosis often begins acutely, and may for a few weeks resemble an attack of acute bronchitis. Two facts should be borne in mind, first that acute bronchitis runs its course in about three weeks, and secondly that it is a bilateral affection, while a beginning tuberculosis is unilateral and almost invariably located at the summit of one or the other lung. It cannot be too strongly emphasized that a cough which, without complications, has persisted for six weeks or more should be carefully investigated; the majority of such cases will be found to be tuberculous. It is worth noting that individuals with tuberculosis not infrequently give a history of being subject to repeated colds.

Acute bronchitis is especially prevalent in the changeable weather encountered in the spring and late autumn. In some instances it is the result of becoming chilled, in others it follows an ordinary coryza, the latter being in many instances an acute infection transferred from person to person. Individuals who are habitually closely confined, or who commonly visit crowded places of amusement, or use public conveyances are especially prone to "acute colds." On the other hand, those who live an outdoor life and are subjected to all sorts of inclement weather are comparatively free from these disorders. The contagiousness of acute colds was noted by Benjamin Franklin, a century and a half ago.

The bacteriological findings in cases of acute catarrhal bronchitis are varied. Occasionally some one organism predominates or even occurs alone, but this is rare. Among the organisms encountered may be mentioned the micrococcus catarrhalis, influenza bacillus, streptococcus and pneumococcus.

An attack of acute bronchitis may be precipitated by the inhalation of dust or chemical irritants, such as ether, chlorine gas, ammonia, etc.

Morbid Anatomy.—Acute bronchitis is practically always a bilateral affection although the distribution of the inflammatory process is not uniform. As a rule, certain portions of the bronchial tree are more affected than others. Not only are the bronchi affected but in most instances the trachea and larynx as well; indeed in some instances the process may be limited to the larynx, trachea and main branches of the bronchial tree (see Fig. 219). When the infection manifests itself principally in the smaller tubes there are apt to be small areas of broncho-pneumonia. The latter may or may not be sufficiently marked to be recognized clinically.

In simple catarrhal bronchitis the mucous membrane is reddened and swollen. It is covered with a sticky exudate which is greyish in color and mucoid in character. In the later stages the exudate often becomes mucopurulent or purulent.

Symptoms.—The onset is frequently with coryza, which as it extends downward, successively causes some irritation of the pharynx, at times hoarseness, often marked in character, and finally symptoms indicating involvement of the tracheal and bronchial mucous membranes, namely, a sense of oppression in the chest, substernal soreness and cough. The latter is dry at first and often occurs in paroxysms which cause marked pain beneath the sternum and lower part of the chest. Fig. 219 illustrates very clearly why this should be so. The expectoration is at first scanty, viscid and difficult to raise. After four or five days the cough tends to become looser, and the sputum becomes, first mucopurulent, later purulent, and is apt to be profuse.

Slight fever, which may range from 100° to 102°F. or higher, occurs in the severer cases. The ordinary case is not febrile for more than a week or so. Later than this the temperature becomes a valuable sign in distinguishing the condition from tuberculosis. Most individuals experience a sense of oppression and languor. Pain in the back and bones, not unlike those encountered in grippe, also occurs with varying intensity.

Physical Signs.- Unless the smaller tubes are involved, as in the aged or very young, there is no increase in the respiratory rate. Aside from the evidences indicative of an acute febrile state, inspection is negative. Percussion shows no changes. On palpation there may be noted a rhonchal fremitus.

The physical signs are almost entirely auscultatory in character. At first the râles are sibilant in character, are heard over both sides of the

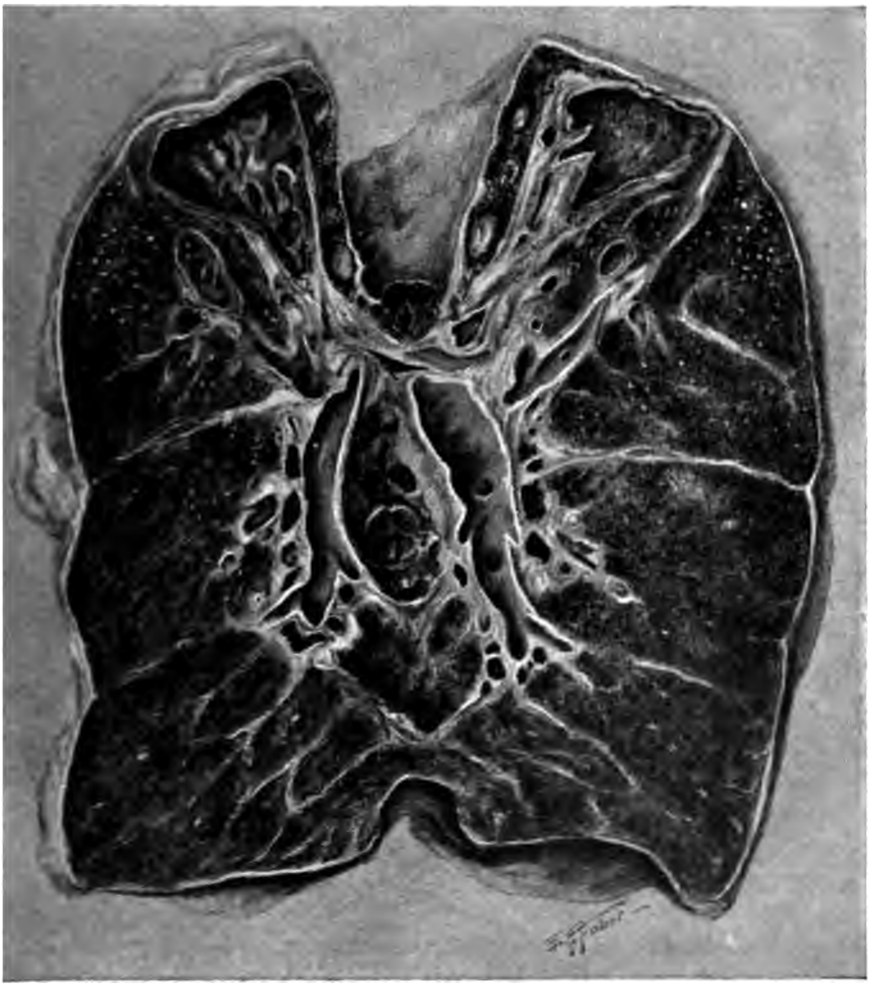


Fig. 219.—Intense acute bronchitis due to influenza bacillus. Upper lobe shows cavity at apex, great thickening of pleura. Adhesions between lobes. (Jefferson Medical College Museum.)

st, and are very changeable, appearing and disappearing, especially r cough. Later as the cough loosens the râles have a moist sound and ubbling quality. While the almost universal rule is that acute nchitis is a bilateral affection, it occasionally happens that the physical s are confined to one side. It is needless to say, however, that such s are unusual and that such a condition is strongly suggestive of erculosis. Until the latter can be ruled out positively, a diagnosis of te bronchitis is indefensible.

Still another variation, and one that is relatively common, is for the mmation to be confined to the trachea alone. In these cases all of symptoms of acute bronchitis are present with an absence of any sical signs in the chest.

A diagnostic error is sometimes committed, owing to the fact that a acute bronchitis occurs in association with an incipient tuberculosis, more serious affection thus becoming masked. Even in those cases re, from the symptoms, one may suspect tuberculosis, the latter is cult to demonstrate until the bronchitis has cleared up. After the ptoms, and especially, the physical signs, of acute bronchitis have ppeared the tuberculous lesion may be detected.

Diagnosis.—The chief features of acute catarrhal bronchitis are za, some irritation in the throat, substernal soreness, cough and ex- oration. For a few days there is usually a slight elevation of the perature. If the process is limited to the trachea and large tubes e will be no physical signs; if the medium-sized bronchi are involved, s will be heard over both lungs. Of itself acute bronchitis is a trivial ction. It is always worthy of consideration, however, as it is often forerunner of more serious trouble. In children and in those of nced years the possibility of the inflammatory process extending to finer bronchi and air cells must always be borne in mind.

In children catarrhal inflammation of the conjunctiva and nasal ous membrane associated with cough and expectoration, may be prodromal symptoms of *measles*. The mucous membrane of the th should be examined for the presence of Koplik's spots. The ptoms in the early stage of *whooping-cough* are, as a rule, those of cute cold, the characteristic whoop not appearing until later. In presence of an epidemic of whooping cough an acute "cold" should be ed upon as possibly being the prodromal stage of pertussis.

Not infrequently patients who develop *lobar pneumonia* will give a ry of having had a "bad cold" for some days before the onset of the monia. Acute bronchitis is so constantly present in cases of *oid fever* as to constitute a symptom of the disease. It may occur in the attack of typhoid fever. In an individual suffering from what ars to be an attack of acute bronchitis and in whom the fever persists prostration is marked, typhoid fever should be thought of.

he differentiation between acute bronchitis and early tuberculosis nsidered on page 326.

CHRONIC BRONCHITIS

tiology.—As a primary affection, chronic bronchitis is encountered less frequently than is usually thought, and while it is commonly ht that repeated attacks of acute bronchitis may develop into the chronic form of the disease, such is rarely the case. Indeed there are some who refuse to recognize the existence of a primary chronic bronchitis under any circumstances. It is certain that many cases so designated are in reality instances in which a much more serious affection is present the bronchitis being only a secondary manifestation There is one class of cases, however, in which chronic bronchitis is very frequently a primary affection, namely, those in which there has been a prolonged exposure to dust, such as is seen in millers, stone cutters, coal miners, etc. In these cases the constant irritation produced by the dust particles, which at first produces a mild acute inflammatory condition of the bronchial mucous membrane, finally brings about a chronic condition. It is worth bearing in mind, however, that the morbidity from tuberculosis is very high among such workers, and care must be exercised not to mistake a latent tuberculosis for chronic bronchitis.

Chronic bronchitis is rarely encountered in the young; on the other hand, it is very frequent among those past the middle period of life. The winter cough so frequently encountered in those of advanced years is usually one of the indications of a faulty circulation, a chronic renal lesion or some chronic pulmonary condition such as asthma or emphysema. Indeed, it is in association with these latter conditions that chronic bronchitis is most frequently met with. In such individuals climatic changes have an important bearing; they are extremely susceptible to sudden changes in the weather. In the warm summer months they are almost entirely free from their pulmonary symptoms, and residence in a warm climate during the winter months also assures them of freedom from their symptoms.

Gout, chronic alcoholism, certain skin lesions and obesity also are not uncommonly accompanied with chronic bronchitis. Rarely a tumor or aneurism of the aorta, through pressure on the bronchi, may bring about a catarrhal inflammation of the bronchial mucous membrane, and finally à chronic bronchitis. It is thus seen that in the vast majority of instances the condition is secondary to some other affection, and until disease of the heart, kidney or other pulmonary lesions are excluded a diagnosis of chronic bronchitis alone is untenable.

Morbid Anatomy.—The condition is characterized by a venous hyperemia and swelling of the bronchial mucous membrane with increased secretion of mucus, and the exudation of serum and pus cells. As the disease progresses the mucous membrane may hypertrophy in places, and in others become atrophied, so that the longitudinal bands of elastic tissue are readily seen. The membrane is also frequently denuded of its epithelium and the glandular tissues atrophied. Of the end results, cylindrical dilatation of the medium and smaller bronchi is frequent, while some degree of emphysema is constantly met with.

Symptoms.—The chief manifestations are as follows: Shortness of breath on the slightest exertion, which is sometimes accompanied by a fit of coughing, with or without expectoration. These symptoms, especially the shortness of breath, are commonly due to the associated emphysema; in some cases cardiac weakness is the exciting factor. The cough, as already stated, is variable, being much influenced by the weather conditions. In the summer it is very slight, or even disappears altogether. Even in the winter it is not apt to be constant, and during this season may occur only in the morning, or there may be a paroxysm at chest, and are very changeable, appearing and disappearing, especially after cough. Later as the cough loosens the râles have a moist sound and a bubbling quality. While the almost universal rule is that acute bronchitis is a bilateral affection, it occasionally happens that the physical signs are confined to one side. It is needless to say, however, that such cases are unusual and that such a condition is strongly suggestive of tuberculosis. Until the latter can be ruled out positively, a diagnosis of acute bronchitis is indefensible.

Still another variation, and one that is relatively common, is for the inflammation to be confined to the trachea alone. In these cases all of the symptoms of acute bronchitis are present with an absence of any physical signs in the chest.

A diagnostic error is sometimes committed, owing to the fact that a true acute bronchitis occurs in association with an incipient tuberculosis, the more serious affection thus becoming masked. Even in those cases where, from the symptoms, one may suspect tuberculosis, the latter is difficult to demonstrate until the bronchitis has cleared up. After the symptoms, and especially, the physical signs, of acute bronchitis have disappeared the tuberculous lesion may be detected.

Diagnosis.—The chief features of acute catarrhal bronchitis are coryza, some irritation in the throat, substernal soreness, cough and expectoration. For a few days there is usually a slight elevation of the temperature. If the process is limited to the trachea and large tubes there will be no physical signs; if the medium-sized bronchi are involved, râles will be heard over both lungs. Of itself acute bronchitis is a trivial affection. It is always worthy of consideration, however, as it is often the forerunner of more serious trouble. In children and in those of advanced years the possibility of the inflammatory process extending to the finer bronchi and air cells must always be borne in mind.

In children catarrhal inflammation of the conjunctiva and nasal mucous membrane associated with cough and expectoration, may be the prodromal symptoms of *measles*. The mucous membrane of the mouth should be examined for the presence of Koplik's spots. The symptoms in the early stage of *whooping-cough* are, as a rule, those of an acute cold, the characteristic whoop not appearing until later. In the presence of an epidemic of whooping cough an acute "cold" should be looked upon as possibly being the prodromal stage of pertussis.

Not infrequently patients who develop *lobar pneumonia* will give a history of having had a "bad cold" for some days before the onset of the pneumonia. Acute bronchitis is so constantly present in cases of *typhoid fever* as to constitute a symptom of the disease. It may occur early in the attack of typhoid fever. In an individual suffering from what appears to be an attack of acute bronchitis and in whom the fever persists and prostration is marked, typhoid fever should be thought of.

The differentiation between acute bronchitis and early tuberculosis is considered on page 326.

CHRONIC BRONCHITIS

Etiology.—As a primary affection, chronic bronchitis is encountered much less frequently than is usually thought, and while it is commonly taught that repeated attacks of acute bronchitis may develop into the

Bronchial casts may be formed secondarily to diphtheria in the pharynx or larynx or to croupous pneumonia. Casts occurring in association with these two diseases will not be considered here.

Etiology.—The cause of fibrinous bronchitis is as yet wholly unknown and there is no evidence to show that it is in any way dependent on a bacterial infection. The disease occurs most frequently between the ages of ten and thirty. Males are affected about twice as frequently as females. In common with other forms of bronchitis this form of the disease occurs more frequently in the colder months of the year.

The immediate exciting causes are probably, in great measure, the same. In one instance a simple bronchitis develops; in the other a fibrinous exudation is later superadded.

Fibrinous bronchitis has been described as occurring in association with a variety of conditions but in most instances the connection is to be regarded as no more than a coincidence. It has been noted in the course of the acute infections such as measles, scarlet fever, erysipelas, typhoid fever, influenza, etc. The inhalation of irritant fumes and gases such as steam, smoke and ammonia, has at times been followed by the expulsion of fibrinous casts. Among other conditions reported may be mentioned asthma, pulmonary edema following thoracentesis, pulmonary actinomycosis and aspergillosis and various skin diseases.

The largest number of instances have been met with in individuals affected with heart disease and pulmonary tuberculosis. In regard to tuberculosis the frequency of the association varies tremendously among different observers. West[1] refers to the association of fibrinous bronchitis and tuberculosis in 7 out of 51 cases. Lehmann-Model[2] found tuberculosis at autopsy in 3 of 6 cases observed by him and refers to 26 autopsies in the literature in which tuberculosis was found in 10. I recall no instance of bronchial casts occurring among the ward patients in the Phipps Institute over a period of thirteen years nor have such casts ever been found in any of the 662 autopsies on tuberculous individuals. Walshe[3] also emphasizes the fact that he had never seen the true disease either in life or in death in an actively phthisical person. It is probably fair to assume that pulmonary tuberculosis plays no part in the formation of the casts and that the association of the two conditions is a mere coincidence. The same may be said of the association with heart disease.

In a certain number of instances the disease occurs in robust, healthy individuals in whom there is no apparent causative factor.

Morbid Anatomy.—The bronchi involved usually represent a circumscribed area but occasionally the disease occurs in a diffuse form. While any part of the bronchial tree may be involved the bronchi of the lower lobes are most frequently affected (West). The process as a rule, commences in the medium-sized bronchi and extends downward. There is little tendency to spread upward. While it is known that the diphtheria bacillus by extension downward, may produce a fibrinous cast in the trachea and large bronchi and that the pneumococcus may lead to the formation of casts in the small bronchi during an attack of pneumonia, there is no evidence to show that fibrinous casts occurring either idiopathically or in association with other conditions, are bacterial in origin.

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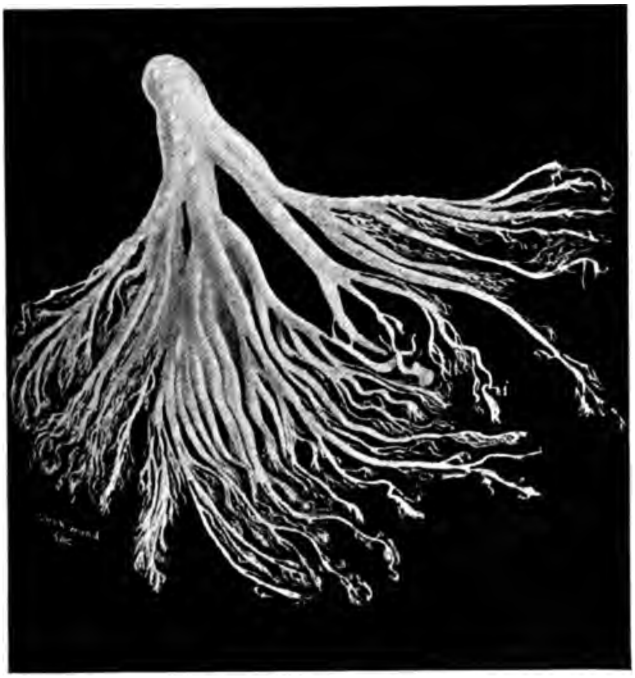
[1] "Diseases of the Respiratory Organs."

[2] Inaugural Dissertation, Freiburg, 1890.

[3] "Diseases of the Lungs," 4th ed., 1871.

to the bronchi themselves there is no constantly associated lesion. asionally a case has been reported in which at the site of the cast, re has been a caseous infiltration, an ulcer or desquamation of the helium. In none of these cases, however, is it clear that the change he bronchi bore any causal relation to the formation of the cast. In chronic form of the disease, emphysema of the lungs sometimes elops.

The casts are pearly gray or white in color and of fairly firm consist- y. They vary in length from fragments ½ inch long up to complete s of 6 or 7 inches, with branches corresponding to the divisions of the nchi from which they have been expelled (Fig. 220). The casts from



IG. 220.—Fibrinous bronchitis cast expectorated; three-fourths natural size. (*Milton Bettmann, in American Journal of the Medical Sciences.*)

larger bronchi are hollow and present a laminated appearance as the ult of successive deposits of fibrin or mucus. The smaller casts are id and often terminate in spirals.

Bronchial casts are usually composed chiefly of fibrin, but in some tances the main constituent is mucus and in others the fibrin and cus occur together in varying proportions. At times the cast is aked with blood. The number expelled varies from one every day o to a large number daily. They may be coughed up as a lump or et or may be surrounded by sputum. Occasionally the cast may be elled in the form of elongated cylinders resembling macaroni or micelli. The discovery of the cast is often accidental. The patient be led to examine the pellet, as in a case recently observed, or the true nature of the small lumps may be revealed in the course of the examination of the sputum. The sputum may be scanty or very profuse. In addition to the presence of casts the sputum may contain Charcot-Leyden crystals, Curschmann's spirals and eosinophilic cells, indicating a condition of the mucous membrane similar to that in asthma (McPhedran).

Symptoms.—Two forms of the disease are recognized, the acute and the chronic. The acute form which is rare, may begin abruptly with a chill, fever, cough, pain in the chest and dyspnea. It is more apt, however, to be associated with one of the acute infections, in which case, after a preliminary bronchitis the paroxysms of coughing become more and more severe and the dyspnea increases. The fibrinous casts, the presence of which alone make the diagnosis possible, may be coughed up at once or after the existence, for some days, of what seems to be a simple bronchitis.

The duration of the acute cases is from a few days to several weeks. In the favorable cases the fever declines by lysis, the cough and dyspnea diminish, the expectoration of casts ceases and there is complete and permanent recovery. In severe cases death often occurs with all the symptoms of suffocation.

Rarely the acute form may become chronic but this is very unusual. The chronic variety is often preceded by the ordinary chronic form of bronchitis although in some instances the disease may assume the fibrinous type from the outset. The attacks tend to recur at certain definite intervals for months or years. In some instances the size of the casts is identical in each attack and this fact in association with the location of the physical signs, points to the involvement of the same portion of the bronchial tree. During the attack the cough assumes a paroxysmal character and dyspnea is marked.

Hemoptysis has been noted in a number of cases. The amount of blood expectorated is usually small and may consist of nothing more than some blood streaks on the cast. Epistaxis, diarrhea and albuminuria have been noted during an attack. In the chronic form there is rarely any fever or any other evidence of constitutional disturbance and the general nutrition is well maintained. Emphysema often develops.

In the chronic form the attack, consisting of paroxysms of dyspnea and coughing followed by the expulsion of a cast, may last from a few days to weeks or even months. The separate paroxysm is usually short although it may be preceded by some hours of coughing. The attacks vary greatly in duration even in the same patient.

Once the chronic form develops the patient is usually liable to attacks the remainder of life. The disease has been noted to recur over a period of twenty-five years. The interval between the attacks varies. In some instances the attacks recur regularly at certain times, as for instance at the menstrual periods; in others it comes irregularly and there may be an interval of years in which the patient is free from the disease.

Physical Signs.—The physical signs do not give definite information. If but a small area is involved they may be entirely normal. In many instances the physical findings are those of acute or chronic bronchitis. If a sufficiently large area is involved there may be deficient expansion on the affected side, a weakened or absent respiratory murmur, dulness and the presence of râles both large and small. These atelectatic areas disappear with the expulsion of the cast.

The abnormal physical signs are commonly found at the bases of the lungs and usually on one side only. The only physical sign peculiar to fibrinous bronchitis is a curious flapping sound beginning in the middle of inspiration and continuing to the end. It is apparently produced by the presence of partially separated casts. Its value is limited, however, as it is present in but a small minority of cases.

Diagnosis.—A diagnosis of fibrinous bronchitis is possible only by the detection of the casts. These may be so surrounded by the sputum as to escape notice. As already stated they may be accidentally discovered by the patient or they may be detected in a routine sputum examination. The condition is to be suspected in patients who give a history of recurring attacks of paroxysmal cough and dyspnea. In individuals seen during the first attack, however, the finding of a bronchial cast is the only criterion.

The following case is an excellent example of the so-called idiopathic fibrinous bronchitis: The patient was a robust, healthy male, aged 47, referred to me because of suspected tuberculosis. The family history was negative and he had never had any illness. Four weeks prior to his coming under my observation he had caught "cold." The trouble started as a coryza and in a few days he developed a severe paroxysmal cough and some dyspnea. For the first two weeks the sputum, which was moderate in amount, was thick and whitish in color, later it had a greenish tinge. On one occasion after a paroxysm of coughing he spat into his handkerchief a small hard pellet which on examination proved to be a cast from one of the medium-sized bronchi. There was a slight elevation of temperature during the attack. The sputum was negative for tubercle bacilli but contained both pneumococci and streptococci. With the exception of one day spent in bed he attended to his professional work, although with some effort. His appetite was good and he had lost no weight.

On physical examination there was deficient expansion at the right base, impairment of the percussion note, suppressed breath sounds, both large and small râles and in addition a "flapping sound" heard at the end of inspiration. The patient was sent to the seashore and when seen two weeks later there was nothing abnormal to be noted over the area previously affected.

The localization of the physical signs, usually at the base of one lung, should serve to differentiate the condition from *simple bronchitis*. The lodgment of a *foreign body* in one of the medium-sized bronchi is apt to give rise to localized physical signs over the lower lobe of one lung but in such cases the evidences of pulmonary mischief are permanent and in addition casts are not expelled in the sputum. Owing to the paroxysmal character of the attacks and the dyspnea fibrinous bronchitis may be mistaken for *asthma*. In the latter condition the physical signs are bilateral and the respiratory difficulty is expiratory in nature. The finding of bronchial casts is the deciding factor in all cases.

SPIROCHETAL BRONCHITIS

In 1906 Castellani[1] called attention to a special form of bronchitis due to spirochetes. Enormous numbers of these organisms may be present in the sputum. Examples of this infection have been observed in India, Ceylon, the Philippine Islands and the West Indies. During the past two years a number of cases have been reported by French, Italian and Belgian Medical officers. It is their belief that the infection was introduced among the allied soldiers by the Asiatic troops.

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[1] *Lancet*, May 19, 1906.

Etiology.—Castellani and Chalmers describe several varieties of spirochetes. The commonest type is a thin, delicate spirochete with numerous small, uniform coils; one of the extremities may be blunt while the other is pointed. A few of the organisms are thick, quite long and with irregular coils, while others are thin and delicate with irregular coils. Spirochetes resembling the *S. refringens* of Schaudinn may also be present. The disease appears to be markedly contagious.

Symptoms.—The disease may occur in an acute and in a chronic form. In the acute form the patient has fever, feels weak, has a cough and usually, scanty expectoration. According to Castellani and Chalmers the condition is not serious. Violle[1] in reporting 30 cases states that the sputa were nearly always red in color and resembled raspberry juice. The disease was relatively benign and had a duration of about a month, but relapses were frequent. Other observers have noted that the sputum, in addition to being hemorrhagic in character, may be intensely fetid.

The chronic form of the disease may result from an acute attack but more often there is no such antecedent history, the affection having a slow, insidious onset. In this type of the disease the patient has a chronic cough, usually most severe in the morning, and the sputum is abundant and muco-purulent in character. The sputum may be blood streaked and at times small hemoptyses occur. The fever does not conform to any definite type. It may be absent, or hectic in type, or it may be present in the morning and disappear in the afternoon. In still other instances fever is present only at irregular intervals. The course of the disease may be prolonged. Castellani has observed one case in which the infection had existed for five years.

The general health may be but slightly affected although some anemia is usually present. Occasionally marked emaciation may occur. The prognosis is, in the great majority of cases favorable.

Physical Signs.—Examination of the chest does not give any very definite information. Little or nothing abnormal may be detected aside from the presence of râles.

Diagnosis.—It is obvious that the condition is most apt to be confused with pulmonary tuberculosis. A spirochetal infection is to be thought of in individuals suffering from a prolonged cough, muco-purulent expectoration and hemoptyses, but in whom neither the tubercle bacillus nor any one of the less familiar organisms can be demonstrated. The possibility of this infection being present may be suggested also by the occurrence of respiratory symptoms in those who have returned from the far East.

BRONCHIOLITIS FIBROSA OBLITERANS

Our knowledge of this condition is of recent origin. Occlusion of the finer bronchi, independent of other marked lesions of the lung, was first described by Lange[2] in 1901. The following year Fränkel[3] reported the first case in which the diagnosis was made during life and a few years later he[1] reported three additional cases.

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[1] *Bul. de l'Academie de Medicine*, June, 4, 1918.

[2] *Deut. Arch. f. kl. Med.*, Bd. lxx, p. 342.

[3] *Ibid.*, 1902, lxxii, p. 484.

Etiology.—In one of Fränkel's cases the condition followed the inhalation of nitrogen tetroxide fumes, in another the inhalation of lime and other dust; in his remaining two cases the cause was not definitely established. Edens[2] observed a case resulting from the inhalation of the fumes from a mixture of hydrochloric and nitric acid. In addition to these cases the causation of which is reasonably clear, similar pathological changes have been reported as having followed measles, whooping cough, syphilis, uncomplicated catarrhal inflammation of the finer tubes and the aspiration of a foreign body. It would seem, therefore, that any condition capable of causing inflammation of the finer bronchi and bronchioles may be followed by occlusion of this portion of the bronchial tree. On the other hand either such an occurrence is rare, except in the case of irritating gases, or it is not generally recognized.

Morbid Anatomy.—In the cases reported by Fränkel and Edens, following the inhalation of a toxic gas, the lungs presented the appearance usually seen after such accidents (see p. 521). In addition the cut surface showed numerous small grayish-white nodules from 1 to 2 mm. in diameter which closely resembled miliary tubercles. On examination with a hand glass these small nodules are seen to differ from miliary tubercles in that they are angular or stellate in form. Futhermore they are found, on dissection, to be the terminal portion of the smaller bronchi. Microscopically the finer bronchi show marked epithelial desquamation, the cells often blocking the tube. In addition there is an ingrowth of connective tissue which may completely occlude the lumen or reduce it to a small slit. The connective tissue apparently takes its origin from the bronchial wall. Connective-tissue invasion of the adjacent alveoli may take place also.

Symptoms.—The dominant symptoms are intense dyspnea and cyanosis. If the inhalation of irritating vapors has occurred there may be present also cough and the expectoration of reddish-brown sputum. The pulse is very rapid. The percussion note is hyperresonant as the result of emphysema and on auscultation numerous fine râles are heard. In one of Fränkel's cases the symptoms developed sixteen days after the inhalation of an irrespirable gas; death occurred three days later. In Edens' case the symptoms developed ten days after the inhalation of gas, death taking place on the twenty-sixth day. It would seem that the appearance of the dyspnea and cyanosis coincide with the subsidence of the acute inflammation and the occlusion of the bronchioles as the result of connective tissue proliferation.

Diagnosis.—In an individual who is known to have inhaled a *poisonous gas* and in whom there is a temporary amelioration or subsidence of the symptoms, a diagnosis of bronchitis or bronchiolitis obliterans may
red if there develops severe dyspnea and cyanosis associated
rresonance and fine râles. If, however, the condition follows
hooping cough, or any condition associated with a catarrhal
ion of the finer bronchi a correct diagnosis would be a matter
cute miliary tuberculosis and broncho-pneumonia may mani-
lves in the same way. In the great majority of cases the

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[1] *Berliner. kl. Woch.*, 1909, xlvi, 6.

[2] *Deut. Arch. f. kl. Med.*, 1906, lxxx, 598.

ractically all cases when the catarrhal stage is well established. The esults obtained by Olmstead and Luttinger[1] in a study of the complenent fixation test in 111 cases of whooping cough or suspected whooping cough also support the claim that the Bordet-Gengou bacillus is the true etiological factor.

Morbid Anatomy.—The characteristic lesion of the disease is found in the trachea and bronchi, especially the former. The opportunity of studying the pathological changes in the acute stage of the disease does not happen very frequently as death is most apt to occur at a later period. In the few cases studied by Mallory and Hornor[2] the microscopic examination showed large numbers of minute bacteria between the cilia of many of the cells lining the trachea. The cilia of single cells or large groups of cells may be affected. The organisms usually reach the base of the cilia but may extend only part way. They frequently cause a lateral spreading or mushrooming of the cilia covering a single cell. In many places the cilia are reduced to short stubs or are entirely wanting. The action of the bacteria appears to be largely mechanical leading to interference with the normal movements of the cilia by sticking them together. In this way the microörganisms furnish a continual irritation.

The most serious of the effects of whooping cough are to be found in the complications. A common cause of death is broncho-pneumonia which is often tuberculous in character. Areas of atelectasis, either alone or in association with broncho-pneumonia, are frequently seen. Enlargement of the bronchial lymph nodes is a common result of whooping cough, and in many cases areas of caseation due to tuberculosis are present. The paroxysms of coughing may lead to over distention of the lungs (emphysema). Among the more unusual complications may be mentioned an acute form of bronchiectasis (bronchiolectasis, see page 296), interstitial and mediastinal emphysema and pneumothorax, the last-named conditions being caused by the rupture of the lung.

Symptoms.—It is customary to divide the clinical manifestations of whooping cough into three stages: (1) The prodromal or catarrhal; (2) the paroxysmal or spasmodic; and (3) the stage of gradual disappearance of the cough and spasm. This division is somewhat arbitrary and does not hold true for all cases. In some instances the disease may begin with violent paroxysms of coughing; in others the characteristic whoop may be wanting.

Between the time of exposure and the appearance of the first symptoms there is a variable incubation period of from seven to ten days. In the *catarrhal stage* the symptoms are those of an ordinary cold. There is slight fever, running at the nose, injection of the conjunctiva and a cough. Even at this time the cough may be spasmodic but, as a rule, it is such as occurs in an attack of simple bronchitis. There is this distinction, however, that the cough is somewhat more frequent and obstinate, both in children and adults, and that the patient has a more troublesome sensation of tickling in the throat and inside the trachea (Trousseau). The catarrhal stage lasts from a week to ten days.

The *paroxysmal stage* dates from the time of the first "whoop." The cough now assumes a definite paroxysmal character. The paroxysm is often preceded by pain beneath the sternum and a sensation of chronic form of the disease, such is rarely the case. Indeed there are some who refuse to recognize the existence of a primary chronic bronchitis under any circumstances. It is certain that many cases so designated are in reality instances in which a much more serious affection is present the bronchitis being only a secondary manifestation There is one class of cases, however, in which chronic bronchitis is very frequently a primary affection, namely, those in which there has been a prolonged exposure to dust, such as is seen in millers, stone cutters, coal miners, etc. In these cases the constant irritation produced by the dust particles, which at first produces a mild acute inflammatory condition of the bronchial mucous membrane, finally brings about a chronic condition. It is worth bearing in mind, however, that the morbidity from tuberculosis is very high among such workers, and care must be exercised not to mistake a latent tuberculosis for chronic bronchitis.

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[1] *Arch Int. Med.*, July, 1915.

[2] *Jour. Med. Res.*, November, 1912.

Chronic bronchitis is rarely encountered in the young; on the other hand, it is very frequent among those past the middle period of life. The winter cough so frequently encountered in those of advanced years is usually one of the indications of a faulty circulation, a chronic renal lesion or some chronic pulmonary condition such as asthma or emphysema. Indeed, it is in association with these latter conditions that chronic bronchitis is most frequently met with. In such individuals climatic changes have an important bearing; they are extremely susceptible to sudden changes in the weather. In the warm summer months they are almost entirely free from their pulmonary symptoms, and residence in a warm climate during the winter months also assures them of freedom from their symptoms.

Gout, chronic alcoholism, certain skin lesions and obesity also are not uncommonly accompanied with chronic bronchitis. Rarely a tumor or aneurism of the aorta, through pressure on the bronchi, may bring about a catarrhal inflammation of the bronchial mucous membrane, and finally a chronic bronchitis. It is thus seen that in the vast majority of instances the condition is secondary to some other affection, and until disease of the heart, kidney or other pulmonary lesions are excluded a diagnosis of chronic bronchitis alone is untenable.

Morbid Anatomy.—The condition is characterized by a venous hyperemia and swelling of the bronchial mucous membrane with increased secretion of mucus, and the exudation of serum and pus cells. As the disease progresses the mucous membrane may hypertrophy in places, and in others become atrophied, so that the longitudinal bands of elastic tissue are readily seen. The membrane is also frequently denuded of its epithelium and the glandular tissues atrophied. Of the end results, cylindrical dilatation of the medium and smaller bronchi is frequent, while some degree of emphysema is constantly met with.

Symptoms.—The chief manifestations are as follows: Shortness of breath on the slightest exertion, which is sometimes accompanied by a fit of coughing, with or without expectoration. These symptoms, especially the shortness of breath, are commonly due to the associated emphysema; in some cases cardiac weakness is the exciting factor. The cough, as already stated, is variable, being much influenced by the weather conditions. In the summer it is very slight, or even disappears altogether. Even in the winter it is not apt to be constant, and during this season may occur only in the morning, or there may be a paroxysm at

Trousseau expressed the opinion that when it is noted that the fits, which have been numerous, suddenly cease, an inflammatory complication is to be suspected.

Physical Signs.—There are no physical signs peculiar to whooping cough other than the change in the character of respiration during the paroxysmal stage. In the catarrhal stage there may be no chest signs whatever or there may be râles as in cases of simple bronchitis. During the paroxysms the percussion note may be slightly elevated and of short duration owing to the diminished air content of the lungs. There is no inspiratory sound and the expiration is imperfectly heard. Following the "whoop" the breath sounds are distant owing to the gradual ingress of the air. A few râles may be present.

Diagnosis.—In the catarrhal stage the diagnosis is not possible although, in the presence of an epidemic or known exposure, the possibility of whooping cough should suggest itself. Catarrhal symptoms also occur as prodromes in measles and as these two infections commonly precede or follow each other both should be kept in mind. In such cases the mouth should always be examined for Koplik's spots.

Once the "whoop" has appeared the diagnosis is easy. In doubtful cases, especially those in which the "whoop" is absent or not characteristic, the complement fixation test may aid in the diagnosis.

When broncho-pneumonia occurs the symptoms and physical signs of that condition dominate the picture. A very serious sequel is tuberculosis. This condition is to be suspected in children who are delicate and in whom fever persists and emaciation and weakness gradually become more and more marked. In these cases the whooping cough serves to arouse into activity a latent tuberculous process already present. Many of the fatal cases of broncho-pneumonia following pertussis are tuberculous in character.

BRONCHIAL ASTHMA

By the term bronchial asthma is meant a form of paroxysmal dyspnea the characteristic feature of which is marked diminution or arrest of the respiratory movements with prolonged expiration. The condition is sometimes referred to as spasmodic asthma. Among the older writers many affections characterized by paroxysmal attacks of dyspnea were referred to as asthma, often having the prefix cardiac, renal, etc. The use of the term asthma should be restricted to the bronchial type of the disorder.

Etiology.—The disease may manifest itself at any *age*, but shows a decided preponderance in favor of the earlier years of life. The following table shows the age distribution in 225 cases collected by Hyde Salter[1]:

	Cases	Per cent
During first year	11	31.0
m 1 to 10	60	
n 10 to 20	30	13.3
n 20 to 30	39	17.3
n 30 to 40	44	19.0
40 to 50	24	1.1
50 to 60	12	5.0
60 to 70	4	1.4
70 to 80	1	0.7

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[1] "Asthma," London, 1868.

In regard to *sex* males are more subject to the disease than females in the proportion of nearly 2 to 1.

It has long been recognized that the condition is often *hereditary*. In a very considerable proportion of cases there is a direct transmission of the predisposition to asthma or hay fever, the latter condition being closely allied to asthma both in its causation and in its clinical manifestations.

The influence of climate is uncertain although in most instances an asthmatic is less subject to attacks in a warm equable region than in one which is cold and subject to marked variations in the temperature. In regard to location the disease often manifests the most curious vagaries. I once knew a man residing in a city not far removed from Philadelphia who suffered severely from asthma and who became free from his attacks on removing to the latter city. In such cases it is probable that some irritant, to which the individual has been susceptible, has been present in one place and not in the other.

In certain instances the asthmatic attacks seem to be related in some way to the presence of nasal polypi, nasal spurs, deviation of the nasal septum or hypertrophy of the turbinates. Very often the removal or correction of these defects is followed by a disappearance of the asthmatic attacks.

It is a matter of common observation that asthmatics rarely develop tuberculosis and most authorities state that asthma rarely occurs in tuberculous individuals. Occasionally, however, tuberculous patients suffer from asthmatic attacks at the onset of or during the course of their malady. I have seen two examples of this.

Exciting Causes.—In practically all individuals who are susceptible to attacks of asthma or *hay fever* there is nearly always some other factor needed to precipitate a seizure. Rackemann[1] classifies the causes of asthma as follows: (1) Extrinsic or those due to the inhalation of horse dandruff, plant pollens, feather dust, etc. (2) Intrinsic or those due to pathological conditions in the nose or throat, uterine disorders, gastro-intestinal disturbances, etc. In the intrinsic group is included a variety of conditions the correction of which has brought about a cure of the asthma. Cooke[2] distinguishes the two groups respectively as (1) Specific or anaphylactic and '(2) nonspecific or not demonstrably anaphylactic.

The effect of the inhalation of various dusts or odors, particularly the odors emanating from animals, is one of the most remarkable features of asthma. In one individual the emanations from the horse provoke a seizure; in another the presence of a cat will give rise to an attack; while in still others rabbits or guinea-pigs are the offending animals. A medical friend previously free from the disease, always developed asthmatic attacks after a visit to the animal room of the laboratory in which he worked. Exposure to the emanations of the animals even for a few minutes, would lead to some difficulty in breathing. Not knowing which animal was the cause of his trouble he made cutaneous tests with the serum of the different animals. The serum from the rabbit gave a slight reaction, while that from the guinea-pig in the course of a few minutes, produced at the site of the inoculation, a marked urticarial swelling. The skin test is also employed in the case of hay fever patients to determine which particular pollen is the offender.

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[1] *Archives of Internal Medicine*, Oct., 1918.

[2] *Medical Clinics of N. Amer.*, Nov., 1917.

The sensitiveness of many individuals to the pollen of various weeds and flowers has long been recognized and in the case of a susceptible individual the inhalation of pollen may give rise to an attack of hay fever or asthma or both. In other instances the inhalation of pungent odors, such as that given off by mustard, will lead to an attack. Finally, exposure to an excessive amount of dust, of whatever nature, will often produce a seizure.

The studies of Walker and his associates at the Peter Bent Brigham Hospital have thrown much light on the cause of asthma. They have shown the frequency with which the asthmatic is hypersensitive to some protein substance. While in some instances the offending protein is readily detected, as for example in the cases of so-called horse asthma, in others the search may be long and devious. Careful study of the patient and his surroundings usually suggests the clue while the skin test completes the incriminating evidence. In 150 cases carefully studied by Walker 55 per cent. were found sensitive to some protein. Of this group horse hair and horse dandruff were the causes in about 20 per cent.; wheat proteins in 15 per cent.; bacteria, staphylococcus aureus, in 15 per cent.; pollens of spring flowers in 15 per cent.; pollens of autumnal flowers in 10 per cent.; the cat in 5 per cent.; etc.—(Fourth Annual Report of Peter Bent Brigham Hospital, 1918). Walker and others have shown that patients are commonly sensitive to the proteins of several types of hair or pollens and in such cases the immunizing treatment should be directed toward the protein which gives the greater degree of sensitization.

While the inhalation of the protein is the most common mode of introduction into the body it has been shown that asthmatic seizures may be caused by the ingestion of certain foods, notably the cereal grains. Walker[1] states that of 20 patients who were sensitive to wheat alone, 15 were relieved of asthma when wheat was omitted from the diet and Turnbull[2] found that individuals sensitive to pollens experienced a diminution of their symptoms by abstaining from bread and boiled cereals. Other articles of food which may be mentioned in this connection are eggs, potatoes, fish and casein.

Hypotheses as to the Cause of Asthma.—Of the many hypotheses which have been advanced to explain the cause of an asthmatic attack but two are worthy of serious consideration, namely: (1) spasm of the circular fibers of the bronchial wall; and (2) a rapid swelling of the mucous membrane of the bronchi. While the first hypothesis is the one most generally accepted as the immediate cause of the attack it is probable that the second factor also plays a part and that both, rather than either one alone, are concerned in producing the phenomena of an asthmatic seizure. Asthma is caused by a disturbance of function, the immediate manifestation of which is a characteristic form of paroxysmal dyspnea. There is no disease of the bronchi or pulmonary tissue, although repeated attacks over a long period of years usually lead to secondary changes.

The most rational explanation of the asthmatic seizure is to be found in the phenomenon of *anaphylaxis* which may be produced experimentally in animals by the injection of an alien proteid. If, for instance, a guinea-pig be given a subcutaneous, intraperitoneal or intravenous injection of normal horse serum, and then, after an interval of ten days or more this injection be repeated, it is found that in the elapsed time the animal has become sensitized, and that while the first injection has been without noticeable harmful effect, the second injection causes a very violent poisoning. Both the symptoms and the anatomical changes resulting from this poisoning are predominantly respiratory in nature. An animal so poisoned will within a minute or so after the second injection, vigorously rub its nose, frequently give a spasmodic sneeze and then begin to breathe rapidly. Quickly following this it is noted that the sides of the chest sink in with each inspiration and finally the respirations become very slow and labored. The animal shortly shows tonic and clonic convulsions, the mucous membranes of the mouth and tongue become bluish and often a spurt of urine is seen. After a brief interval in which respiration ceases entirely the breathing is resumed, the respirations being slow and causing but little movement of the chest; gradually the respirations become weaker and weaker and finally cease entirely. Auer and Lewis state that the characteristic feature of anaphylaxis, as seen in the guinea-pig, is extreme distention and immobilization of the lungs. Anatomically the following condition is found:

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[1] *Archives of Internal Medicine*, Oct., 1918.

[2] *Boston Med. and Surg. Jour.*, Oct., 1918.

"The diaphragm is much less arched than in normal animals after death. On opening the chest the lungs present a striking sight; the lungs do not collapse, as normal lungs do when the thoracic cavity is opened, but remain almost fully distended. They look pale bluish pink, and apparently form a cast of the thoracic cavity. Even when excised *in toto* there is practically no collapse and the posterior surfaces often clearly show the markings of the ribs. The lungs are light, soft and spongy and float on water like a cork. On cutting away pieces of lung tissue these pieces do not collapse, but remain distended; the cut surface is usually dry and on pressure a good amount of air may be expressed. Occasionally this pressure reveals some small foci of white foam, as if there were beginning pulmonary edema; occasionally small hemorrhages were seen on the surface of the lungs. The trachea and bronchi usually were dry, but showed often a marked congestion of the mucosa."

The explanation of this extreme inflation of the lungs in association with the difficulty in breathing is to be ascribed to a tetanic contraction of the muscles of the finer bronchioles so that the air is imprisoned in the alveolar sacs. Auer and Lewis state that the anatomical basis for this is clear and that the condition can be easily recognized. The finer bronchioles are practically nothing but muscular tubes and muscle fibers are also present in the alveolar ducts. The contraction of their structure, therefore, must have a profound effect upon the volume of air passing to and from the alveoli and the condition may be still farther aggravated by submucous edema of the bronchi. The opportunity to examine the lungs of an individual who has died during an asthmatic attack rarely occurs so that it is not possible to compare the conditions found in man and experimental animals from the anatomical standpoint. We do know, however, that in man both the symptoms and physical signs point strongly to marked over-distention of the lungs; that the air is taken in with difficulty and expelled with even greater difficulty; and that the condition abruptly ceases either spontaneously or often as the result of an injection of atropine. Everything points, therefore, to spasm of the bronchioles as being the immediate cause of the phenomenon.

Taking up next the exciting cause of the attack it will be recalled that in considering the etiology we pointed out that an asthmatic seizure or an attack of hay fever was often precipitated by the inhalation of some form of dust or animal odor. In the anaphylactic phenomenon as seen in animals, "The reaction of intoxication would seem to be a cellular one, dependent upon a heightened power of assimilation on the part of cells which have been subjected to the anaphylactic substance over a definite period of incubation" (Gay and Southard). As a general rule we have no knowledge of how man has become sensitized to various substances, exposure to which induces an attack. That sensitization has taken place, however, there can be no doubt as contact with certain animals, the inhalation of certain pollens, etc., promptly induces in some individuals, an attack of asthma or hay fever or both. Attacks which are induced by overeating or constipation may also be explained on the ground of proteid intoxication. In those instances in which attacks occur in one locality and not in another it is probable that the particular substance to which the individual is sensitive is present in one place and not in another. This assumption is amply borne out by the fact that hay fever patients either escape an attack entirely or have their symptoms greatly modified by residence in a locality which is free from the pollen to which they are especially susceptible.

Finally, we have to consider the question of heredity upon which much stress has been laid. It has become traditional to ascribe to the majority of individuals who suffer from asthma or hay fever, a neurotic tendency. This hypothesis it seems to me has no real basis in fact and to perpetuate it is a mistake. If we accept the theory that the primary cause of asthma or hay fever be a cellular hypersensitiveness to certain substances, it can easily be seen that this sensitiveness may very readily be transmitted from parent to child. And here again the experimental data may be applied to the disease as seen in man. Thus it has been shown in animals that if the female be sensitized to an alien proteid, such as normal horse serum, susceptibility to this proteid is transmitted to her offspring.

Admitting that there are instances in which it is not possible to determine the exciting cause and admitting also that an asthmatic attack rarely if ever ends fatally it must be conceded that the fulminant type of respiratory distress which may be produced experimentally in animals bears a close analogy to the asthmatic seizures which occur in man.

The so-called anaphylactic reactions of asthma, etc., are commonly regarded as exceptional and pathological events, but we must suspect a qualitatively similar, if quantitatively different, biological response whenever the sensitized being breathes air containing the appropriate
[redacted] vall).

-The essential feature of asthma is a paroxysmal attack
ie attack may occur with explosive suddenness or it may
premonitory symptoms. The latter are varied in charac-
in about one-half of the cases. An individual subject to
ften predict an attack because of drowsiness, neuralgia,
y or sneezing. In other instances the attack is pre-
cy or marked diuresis. I have already referred to the

rôle played by odors, exposure to which will bring on a seizure within a a few minutes.

The first evidence of the attack itself is a feeling of tightness or oppression in the chest. This may be present for a day or so or it may manifest itself suddenly. The attack may occur at any time but it has long been noted that it is apt to manifest itself in the night. The patient goes to bed feeling perfectly well, when he is suddenly awakened by a feeling of suffocation or constriction about the chest. The difficulty in breathing rapidly increases and often reaches an extreme degree. The face is pale and anxious and may be covered with a cold perspiration. Owing to the deficient oxygenation of the blood the lips are usually of a dusky hue and in a severe attack the face may also be of a leaden or dusky color. The eyes may protrude and the nostrils may be dilated. In the effort to obtain more air the patient may rush to an open window. More often, however, he assumes a fixed position. This may be standing up with the hands grasping some support or sitting up in bed with the hands pressed upon the bed. In either case the object is to give an additional purchase to the accessory muscles of respiration. The head may be thrown back or it may be thrust forward and sunk between the shoulders, "turtle-like."

The mechanical difficulty in asthma is in expelling the air from the lungs. The lungs are greatly distended and owing to the spasm of the finer bronchi the air vesicles are incapable of emptying themselves. The inspirations are reduced in frequency, sometimes to one-half the normal. They are jerky in character and much shortened in duration, the normal ratio between inspiration and expiration being in extreme cases, reversed. In spite of the desperate efforts on the part of the patient the chest scarcely moves because the lungs are already fully distended. The supraclavicular and suprasternal depressions are more pronounced and the intercostal spaces are sunken.

During the attack the pulse may be small but usually it is but little affected and the temperature is normal.

The duration of an attack may be from 10 or 15 minutes to several days, but when prolonged beyond several hours the severity of the symptoms is usually diminished.

There is no disease in which during the height of an attack, the symptoms are so distressing and alarming and yet at the same time are so free from danger. Death rarely, if ever, occurs as the result of an attack of spasmodic asthma.

There may be a short dry cough at the onset but during the height of the attack, cough is inconspicuous. As the paroxysm subsides expectoration usually begins and this is attended with a cough. The sputum at first occurs in the form of small grayish pellets. Later it gradually becomes more and more copious and also changes from the tenacious mucous to a thin frothy material. In very violent attacks the sputum may be blood-streaked. Any considerable amount of blood, however, is probably an indication of some associated pulmonary lesion. Microscopically the sputum is seen to contain many eosinophiles, Charcot-Leyden crystals and Curschmann's spirals.

I have already referred to the close relationship which exists between bronchial asthma and hay fever. Individuals who are subject to hay fever as a rule suffer no inconvenience except during the fall of the year when exposure to various pollens is common. In the majority of hay fever patients the symptoms are those of an aggravated attack of coryza. In addition they are apt to be subject to sneezing, a paroxysmal cough and considerable depression. Disturbance of the respiratory function varies greatly. It may consist of nothing more than a slight feeling of constriction or it may be indistinguishable from true asthma. Again, in the same individual the coryza symptoms may predominate one year while at another time the asthmatic symptoms are the most severe. Occasionally the inhalation of the pollens will give rise to acute gastro-intestinal disturbances in addition to hay-fever-like manifestations. An example of this is reported by de Besche.[1]

Paroxysmal attacks of sneezing are also to be looked upon as a minor manifestation of irritation of the finer bronchi. Finally, there is to be considered *urticaria* which usually occurs alone but may be associated with asthma. It will be recalled that urticaria is a well-recognized anaphylactic phenomenon and frequently is seen after the injection of a foreign proteid. Within the past few years the attempt has been made to show that *eczema* is a manifestation of anaphylaxis but the evidence so far assured is far from conclusive.

Attacks of bronchial asthma may occur at intervals for years without producing changes in the bronchi or pulmonary tissue. Cases of this type are, as a rule, those which arise as the result of exposure to some irritant such as the pollen of flowers or weeds or animal odors. In many instances, however, the attacks occur early in life and tend to increase in severity. Under these circumstances the individual becomes definitely "asthmatic." Both the severity and the duration of the seizures gradually bring about permanent over-distention of the lungs so that in addition to being subject to attacks of spasmodic dyspnea the individual becomes emphysematous.

Physical Signs.—*Inspection.*—The general features of an attack of asthma have been dealt with in describing the symptoms. The physical appearance of the patient will depend largely on the duration of the disease. If chronic and of long standing the chest, as a result of the associated emphysema, will present the characteristic barrel shape of the latter disease. On the other hand, those who suffer from asthmatic attacks only occasionally, or who have but recently developed the disease, will show no change in the contour of the chest.

During an attack there are two striking features to be noted by inspection: (1) The over-distention of the chest, and (2) the very slight expansile movement of the thorax. This is in striking contrast to the forcible action of the accessory muscles of respiration. The diaphragm is depressed and the abdominal muscles rigid. During inspiration, which is short and quick, the intercostal spaces are drawn in. In most cases the prolongation of expiration is apparent on inspection.

Palpation.—This confirms the deficiency of the expansible movement
oted on inspection. Vocal resonance may be normal but
a severe one it may be greatly diminished partly because
tended lung and partly because of the inability of the
k loudly.

-On percussion the note will vary from that which is
hich is markedly hyperresonant. The degree of exaggera-

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[1] *Jour. Infect. Dis.*, 1918, 22, p. 594.

tion of the resonant quality of the percussion note depends on the amount of associated emphysema. If the latter condition is a prominent feature the percussion note will be hyperresonant all over the chest and the area of cardiac dulness absent.

Auscultation.—During the height of the attack the vesicular murmur is usually inaudible and instead a to-and-fro wheezing sound is heard. As the attack begins to subside innumerable râles are heard throughout the chest. At first the râles are sibilant and sonorous in character and may be both low- and high-pitched. Later fine moist crepitant and subcrepitant râles are heard, especially over the lower portions of the lungs. The short, jerky character of the inspiratory effort and the prolongation of the expiratory act are plainly evident on auscultation. The vocal fremitus may be normal or exaggerated, but as a rule the distress of the patient is such that the use of the voice is not attempted.

Diagnosis.—The term asthma should be restricted to that form of spasmodic dyspnea in which the chief feature is the difficulty in emptying the lungs, that is, it is an expiratory form of dyspnea. In all other forms of dyspnea the principal difficulty is in getting air into the lungs. In those instances in which there is an obstruction in the upper respiratory tract there may be trouble both in inspiration and expiration. Obstruction most commonly occurs in the larynx or trachea and may be brought about by edema of the larynx, diphtheria, inflammatory swelling of the larynx due to some irritant or the obstruction may result from pressure without as by tumors, enlarged lymph nodes or a thoracic aneurism. In such cases the dyspnea is usually quite plainly inspiratory in character and is also apt to be accompanied by a stridor. Aphonia due to paralysis of the vocal cords may occur also. These cases, as a rule, offer no difficulty, but in an individual suffering from the first asthmatic attack, the various causes of dyspnea should be borne in mind.

Cases of *chronic heart disease* frequently suffer from dyspnea. In some instances the dyspnea may appear suddenly and in a paroxysmal form. The inspiration is free, and the expiration is not prolonged. Examination of the heart will give the correct clue to the origin of the trouble. Attacks of paroxysmal difficulty in breathing which appear first after middle life are quite commonly cardiac in origin. In some of these cases the dyspnea is enhanced by the coincident presence of acidosis.

Attacks of dyspnea, sometimes characterized by a sudden onset are not infrequent in cases of *nephritis*. The presence of edema, a high blood-pressure, a ringing second aortic sound and the urinary findings serve to establish the true nature of the dyspnea.

Having established the fact that the case is one of true bronchial asthma it is equally important to determine, if possible, whether the exciting cause is a protein and if so the particular protein for on this fact depends, to a great extent, our ability to relieve the patient. In the case of hay fever or of asthmatic attacks obviously related to exposure to flowers or weeds, skin tests made with the extracts of the pollen from various plants will usually indicate the one to which the patient is alone or most susceptible. If an animal protein is the offender the history of the case will usually indicate its source. Of the food proteins it is to be borne in mind that wheat is the most common offender. In those instances in which the more common protein cannot be implicated a reful study should be made of the patient's environment, especially regards dust exposure and also his dietary habits.

BRONCHIECTASIS

In individuals who have suffered from one of the various inflamatory affections of the lungs or bronchi some degree of bronchial dilation is not an infrequent finding in the autopsy room. The usual aching is that clinically bronchiectasis is relatively uncommon. This, believe, to be a mistake. The currently accepted view that bronchiecasis is always characterized by large quantities of very foul smelling putum and that it is almost invariably an affection of the lower lobes, ust be revised. As a rule, only those cases are recognized, in which he condition has become distinctly apparent and in which the symptoms re typical; and even in these cases the true nature of the trouble is ften overlooked in the belief that tuberculosis is present.

Etiology.—Dilatation of the bronchi is practically always a secondary ffection and may be traced to some preceding disease of the bronchi, ungs or pleuræ. In the great majority of cases of bronchiectasis the ondition is chronic and its evolution gradual. Occasionally it is met vithin an acute form. (See article on Influenza.)

In considering the exciting factors the classification suggested by owler[1] is the most satisfactory.

A. Intrinsic, or conditions acting directly through the bronchi.

1. Bronchitis, acute or chronic. The chronic form of bronchitis is most likely to lead to bronchiectasis as the long-standing inflammatory rocess tends to weaken the bronchial wall. As the result of severe ttacks of coughing the weakened wall is unable to resist the pressure of ir and hence the bronchi dilate. Another factor is the mechanical plugging of the bronchioles by the catarrhal secretions. A slight degree of dilatation of the bronchi is not infrequently met with in cases of emphysema and asthma owing to the fact that those two pulmonary affections are almost constantly associated with a chronic bronchitis.

In my experience one of the most frequent causes of bronchiectasis s the prolonged exposure to *inorganic dust*. At first the dust produces an rritation of the mucous membrane of the upper respiratory tract. Later t induces a subacute inflammation of the bronchial mucous membrane nd in addition leads to connective tissue proliferation in the interlobular epta. Of 21 potters whom I have had under observation the evidences f dilatation of the bronchi were present in 10. Dilatation of the ronchi has also been noted very frequently in cases of anthracosis.

Of the microörganisms, the influenza bacillus seems to be the most nportant as a causative agent.

2. Narrowing of the lumen of the bronchus. This may be caused y a gumma, a foreign body, or by pressure from without as in the case an aneurism or a tumor. As the result of the stricture the bronchial cretions accumulate, changes in the bronchi and peribronchial tissues ke place and the surrounding pulmonary tissue becomes indurated. hese conditions tend to weaken the bronchial wall and if there be added olent expiratory efforts dilatation ensues.

Chevalier Jackson[2] has emphasized the importance of bearing in mind that a *foreign body* is often the cause of bronchiectasis. For this reason, all cases of bronchiectasis of the lower lobes should have an X-ray examination made.

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[1] FOWLER and GODLEE: "Diseases of the Lungs," 1898.

[2] *Penna. Med. Jour.*, August, 1916.

B. Extrinsic, or causes external to the bronchus.—While dilatation can occur as the result of changes in the bronchus alone the condition is more apt to develop if there are, in addition, associated changes in the pulmonary tissues.

(*a*) *Tuberculosis.*—In a very considerable number of cases of chronic ulcerative tuberculosis some dilatation of the bronchi is encountered, although this is usually not recognized clinically. The more chronic the tuberculosis and the greater the amount of fibrous tissue present, the more certain is there to be some evidence of bronchiectasis. In 197 consecutive cases autopsied at the Phipps Institute the bronchi were noted as being dilated in the upper lobes in 20 instances; in the middle lobe of the right lung 9 times and in the lower lobes 9 times. In 6 additional cases the dilatation was sufficiently marked to constitute a true bronchiectasis; of this number 4 occurred in the lower lobes.

(*b*) *Syphilis* may cause dilatation of the bronchi in one of two ways: (1) In the form of a gumma obstructing the lumen of the bronchus. This has been considered among the intrinsic causes. (2) The occurrence of pulmonary fibrosis as the result of syphilis. L. A. Conner[1] in a study of syphilitic stenosis of the trachea and bronchi found that bronchiectasis did not occur in more than 20 per cent. of the cases. Just as in tuberculosis and other chronic pulmonary affections he considered that "much more potent factors are the changes occurring in the bronchial wall and in the adjacent lung tissue, by which the muscular and elastic tissue of the bronchi, upon which their strength and resilience depend, is replaced by inflammatory tissue."

(*c*) *Compression of the Lung.*—The most frequent cause of compression of the lung is a pleural effusion, either serous or purulent in character. If the effusion is unrecognized and the lung remains compressed for some time a proliferation of connective tissue takes place in the collapsed lung and the bronchi also dilate. It is probable that the dilatation occurs, more as the result of the distending force of the cough, acting on bronchi deprived of their normal support, rather than the associated fibroid changes in the lung.

(*d*) *Croupous and Broncho-pneumonia.*—Although delayed resolution following croupous pneumonia is frequently given as one of the exciting causes of pulmonary fibrosis and bronchiectasis, there is some question as to whether such a thing as "delayed resolution" ever occurs. If it does it is extremely rare. In the vast majority of instances what is taken for delayed resolution is in reality an effusion. As a result the lung becomes compressed and if the compression is exerted sufficiently long, pulmonary fibrosis and dilatation of the bronchi ensue.

Bronchial dilatation, in some instances, apparently originates in an attack of broncho-pneumonia. Under these circumstances the dilatation seems to arise partly as the result of the associated inflammatory condition of the bronchi and adjacent alveoli and partly as the result of collapse of air vesicles adjacent to the pneumonic process. As a rule the collapsed vesicles become reëxpanded after recovery from the acute ck. If, however, the change is permanent the atelectatic areas ergo a fibroid change and the smaller bronchi dilate.

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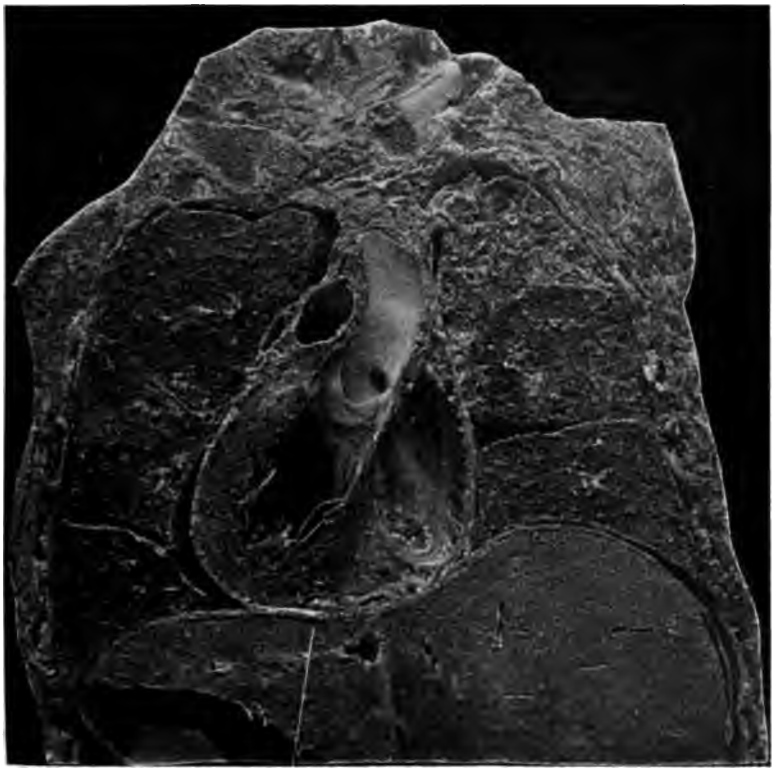
[1] *Am. Jour. Med. Sc.*, 1905, vol. cxxx.

e) *Fibrosis of the Lung.*—This will be considered in detail under ther heading. It may be stated here, however, that in practically ry case in which there is an overgrowth of fibrous tissue in the lungs, re is also dilatation of the bronchi.

(*b*) *Inflammation of the Pleura.*—Marked thickening of the pleura frequent finding in cases of bronchiectasis. Some observers hold the w that a chronic adhesive pleurisy is one of the factors capable of nging about dilatation of the bronchi, but this is difficult to prove.



ı. 221.—Fibrosis of the right upper lobe with dilatation of the bronchi. Such a case would give the signs of a cavity.

asmuch as the two conditions not infrequently occur independently each other, it is quite probable that when they are associated they ve nothing in common.

A consideration of the above etiological factors shows that while eakening of the bronchial wall is the essential feature in all cases that, a rule, one or more additional factors are at work in any given case. esides disease of the bronchial walls, therefore, the effect of increased trapulmonary pressure, the pressure of accumulated secretions within e bronchi, and various chronic affections involving the parenchyma of e lung, are also to be taken into consideration.

Morbid Anatomy.—Although at the autopsy table a large number of the tubes are, as a rule, found to be affected, the disease in the early stage is much more limited. The successive involvement of previously healthy tubes is brought about by the inhalation of the secretions, intercurrent attacks of bronchitis, septic broncho-pneumonia and frequent cough. The condition may be limited to one lung or both may be affected. In 35 cases seen in the Brompton Hospital, Fowler states that in 23 cases (or 65 per cent.) both lungs were affected; the lesion was limited to one lobe in only 8. In 52 autopsies Lebert found that the condition was limited to one lung in 52 per cent. and that in 48 per cent. both were involved. It is usually believed that the lower lobes are most commonly involved and that localized dilatation of the bronchi in the apices of the lungs is unusual. Lebert, however, found unilateral upper lobe involvement in 11 per cent. of his cases. In chronic non-tuberculous affections bilateral involvement is the most frequent while in tuberculous cases unilateral and apical involvement is the most common. Apical involvement is also quite common in cases of pneumoconiosis. McCrae and Funk[1] have reported 5 cases of apical bronchiectasis, 4 of which were associated with tuberculosis (see Fig. 222).

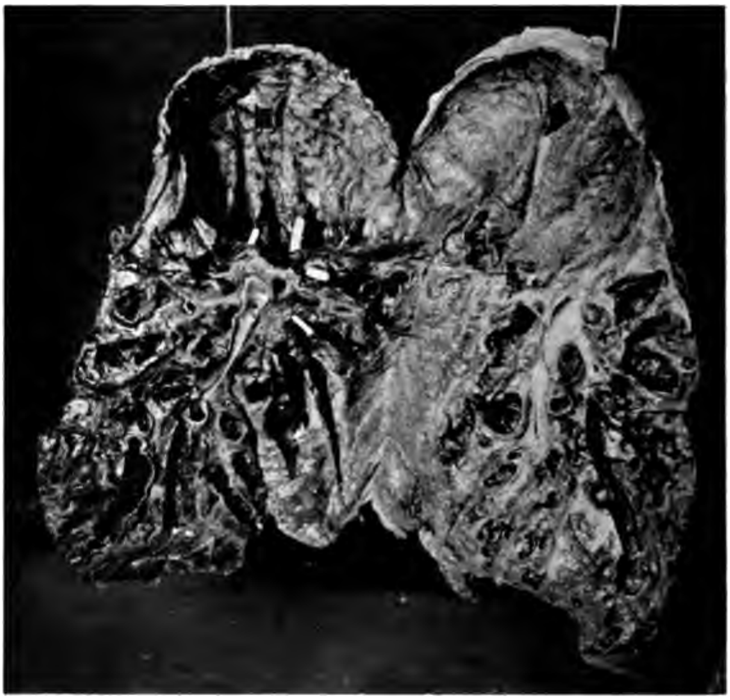


FIG. 222.—The bronchiectatic cavities have been laid open, revealing their wide extent and intercommunication. (*Jefferson Medical College Museum.*)

Two main varieties of bronchiectasis are recognized, namely, the cylindrical and the globular or sacculated.

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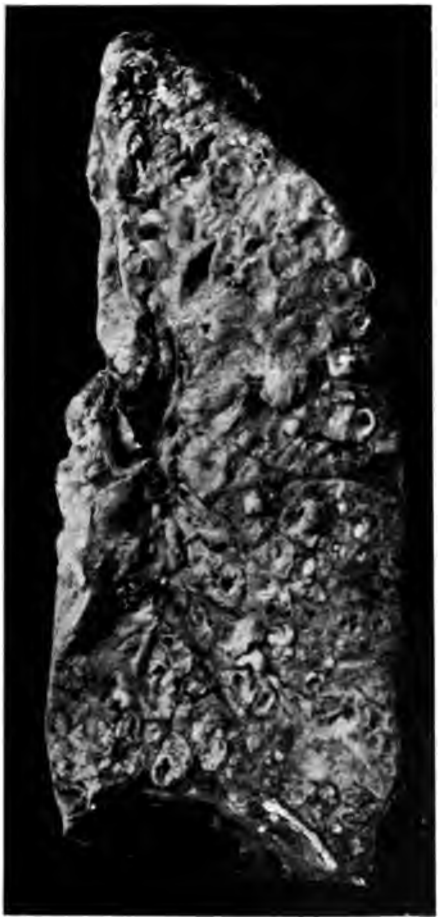
[1] *Jour. Am. Med. Assoc.*, Oct. 7, 1916.

the *cylindrical form* the dilatation is uniform, the smaller bronchi
ıng the size of the larger division instead of diminishing in diameter.
ronchi tend to progressively increase in size toward their terminal
ıities, thus giving rise to an appearance resembling that of the
s of a glove (Wilson Fox). This type of the disease is well illustrated
. 222. A subvariety of the cylindrical form is the fusiform type, in
the dilated bronchi taper somewhat toward their terminal extremity.



24.—An unusual example of fibroid phthisis with extensive dilatation of the bronchi. (*Pennsylvania Hospital.*)

he *sacculated type* shows the most extreme degree of dilatation.
is form the tube may dilate at a single point, forming a pseudo-
y. A number of these may occur in one tube, the caliber of the inter-
g portions remaining approximately normal. The sacculation
ınvolve only one side of the tube, the remainder of the circumference
ıing its normal shape. A subvariety of the sacculated form is
n which the affected bronchus presents a number of bead-like dila-
ıs which have been likened to a rosary (Fox).

The cylindrical type of dilatation is usually found in the larger and medium-sized tubes. While the sacculated type may occur in the larger tubes, it is most commonly encountered in the terminal bronchi. All types of dilatation may be seen in one lung.

The largest and most extensive bronchiectases are encountered in lungs which have undergone marked fibroid changes (see Figs. 223 and 224).

Associated Changes in the Lungs.—The pulmonary changes have been very tersely expressed by Walshe as follows: "The surrounding tissue is either slightly condensed by pressure, hardened by chronic pneumonia, rarefied by emphysema or perfectly natural." A septic broncho-pneumonia due to inhalation of the secretions is not an infrequent terminal event and occasionally gangrene of the lung occurs as the result of perforation of the bronchial dilatation.

Changes in Other Organs.—Dilatation of the right heart may occur as the result of the obstructed circulation in the lungs. Owing to the stagnation of septic material in the bronchial tubes the absorption of toxins frequently leads to amyloid change in the liver and kidneys. Abscess of the brain not infrequently has its origin in a bronchiectasis.

Changes in the Extremities.—One of the peculiar features of the disease is the remarkable change which takes place in the extremities. While clubbing of the fingers and toes occurs in other conditions, it is seen in its most extreme form in bronchiectasis. And the same is true of hypertrophic pulmonary osteoarthropathy. These two conditions, which are almost exclusively associated with diseases of the lungs, will be considered under a separate heading.

Symptoms.—It can be readily understood that the symptoms vary greatly in different cases. In many instances the symptoms are so slight or are so masked by associated changes in the bronchi or lungs that the presence of bronchiectasis is not even suspected. Keeping in mind the various conditions with which the condition is commonly associated will often lead to a correct diagnosis. In a small proportion of cases the diagnosis can be made from the presence of two symptoms, namely, a paroxysmal cough which may occur but two or three times a day and the expectoration of large quantities of purulent sputum which may, in some cases, be horribly fetid. *Coughing paroxysms* are most marked in the morning and again at night when the patient lies down This often serves to empty the cavities and it is not until they again fil up that the paroxysm is repeated. The sputum may be raised easily o it may be brought up only after a severe coughing attack.

The sputum, which may amount to 20 ounces or more in 24 hours, i usually yellowish in color and on standing separates into three layer The lower layer is opaque and grayish in color; the middle consists of thin, turbid fluid, and on top of this is a frothy, brownish-colored laye

In cases of long duration the sputum often has a horribly fetid odo There is probably no disease in which the sufferer becomes such an o ject of aversion to his family as bronchiectasis. I recall one patient, lad of seventeen, who practically became an outcast because of h affliction. In this case the whole house became permeated with t gangrenous odor of his sputum after a coughing attack. In another ca in which the bronchiectatic cavities were emptied during the admin

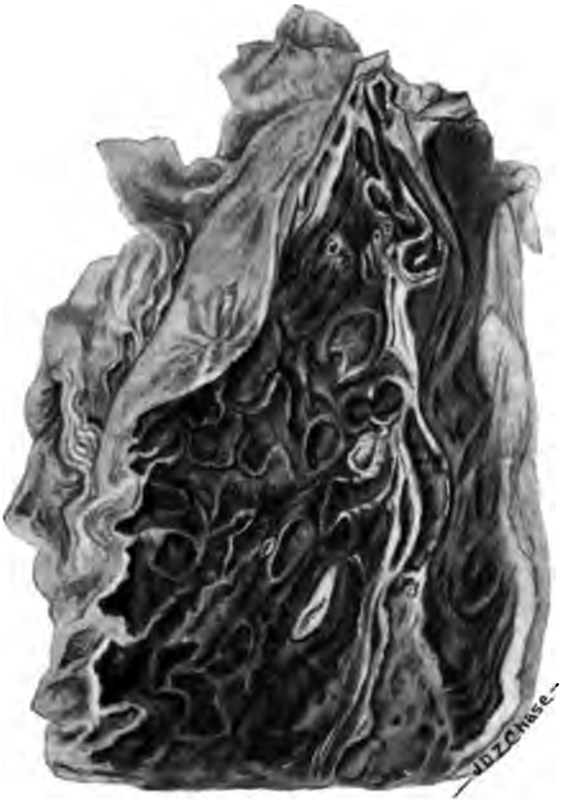


Fig. 223.—Carnification of the lung with dilatation of the bronchi as the prominent feature. This lung on section presented the appearance of a series of coalesced rings measuring from 5 to 15 mm. in diameter. (Case No. 4702, Phipps Institute.)

tration of an anesthetic, the odor was so nauseating that several of the onlookers vomited at once.

Sputum of the type described is not by any means the rule, however. Among a very considerable number of cases of bronchiectasis I have seen there were but four or five in which the sputum was fetid. It is rarely so if the bronchiectasis is confined to the apices as the drainage is freer and the secretions do not stagnate in the bronchi.

Blood-streaked sputum and even small hemoptyses are of not infrequent occurrence and occasionally death is produced by a large pulmonary hemorrhage. The smaller hemorrhages are due to ulceration of the bronchial wall; the larger ones to an erosion of a branch of the pulmonary artery. *Dyspnea* is usually a prominent symptom when the disease becomes well established. It may be due to a variety of causes; dilatation of the right side of the heart, emphysema, extensive fibrosis of the lung, fixation of the diaphragm, etc.

Pain may occur during the course of the disease from extension of the pleural inflammation.

As the disease progresses circulatory disturbances are apt to become more and more marked until finally all the evidences of failing compensation develop.

In spite of the large amount of septic material which is more or less constantly stagnating in the bronchi, *constitutional symptoms* are not marked. As a rule cases of bronchiectasis are afebrile although they are apt, at times, to have periods of fever due to intercurrent attacks of bronchitis or broncho-pneumonia as the result of the invasion of a new area of bronchial or pulmonary tissue. During these attacks the cough is more severe and night sweats commonly occur.

For varying periods of time the general health is little if any impaired but as time goes on there is a gradual deterioration in strength and nutrition and finally death ensues as the result of cardiac failure, general cachexia, or some terminal infection.

Physical Signs.—*Inspection.*—Extreme clubbing of the fingers is so commonly associated with dilatation of the bronchi as to be of decided value in diagnosis. The toes may be clubbed also and in some instances the end of the nose becomes bulbous in appearance.

Owing to the fact that dilatation of the bronchi is secondary to such a variety of diseases and the changes produced by these diseases are so diverse in character, the physical signs vary tremendously. In some instances the bronchiectasis is deep seated and gives rise to no signs whatsoever. A case of this type was seen recently at the Phipps Institute. The patient complained of cough and expectoration but the examination of the chest failed to reveal any abnormality. Several weeks later the patient died as the result of a brain abscess. At the autopsy deep-seated dilatation of the bronchi was found in the lower lobe of the right lung.

The cases which most frequently escape detection are those in which the affection is bilateral and associated with chronic bronchitis, emphysema, or diffuse bilateral fibrosis of the lungs; or in which tuberculosis and bronchiectasis coexist. In these cases the emphysema or tuberculosis tends to mask the bronchial affection. The condition may be suspected if marked clubbing of the fingers is present, in what seems to be a case of emphysema, or if cavity signs are more marked in the lower lobes.

In the bilateral cases, the chest shows no discrepancy between th two sides; it may be normal in appearance or of the emphysemato type.

The type of bronchiectasis which is most frequently recognized is th which involves a part or all of one lung. In these cases there is marke retraction of the affected side and expansion is greatly diminished entirely absent. The heart is usually displaced towards the affecte side.

Palpation.—If emphysema is present the tactile fremitus is reduce In unilateral involvement associated with pulmonary fibrosis the fremit is increased.

Percussion.—The character of the percussion note will depend almo entirely on the nature of the associated pulmonary changes and th location of the dilatations. In deep-seated bronchiectases without an change in the parenchyma of the lung, the note is normal; if emphysem is present, it is hyperresonant, and if consolidation has occurred as th result of a chronic interstitial pneumonia, the note will be dull.

When the disease is confined to one side the opposite lung is usuall hypertrophied as the result of compensatory emphysema. This is in dicated by the fact that the pulmonary resonance extends beyond th middle line anteriorly; in addition loud puerile breathing will be hear on this side.

Superficially placed dilatations will give rise to a high-pitched tym panitic note. If the dilatations are numerous a distinct tympaniti quality may be lacking but will influence the other percussion change by raising the pitch. In unilateral cases the area about the angle of th scapula is where one is most apt to elicit a tympanitic note.

Auscultation.—The auscultatory signs present the greatest variation If the condition is bilateral the respiratory sounds may be identical wit those heard in emphysema or they may be slightly suppressed with bronchovesicular quality, especially during expiration. In the unilater cases associated with fibrosis, the breath sounds are, for the most par suppressed but in certain areas are definitely cavernous. The latt type of breathing is very frequently heard at the angle of the scapul Over the area in which cavernous breathing is heard bronchophony an whispering pectoriloquy may be marked. *Skoda's veiled puff* is ofte referred to. This sound occurs at the end of inspiration and sounds though a puff of air were entering a cavity situated just beneath the ea In my experience this sign is not often present.

Râles of every description may be heard. When coarse and metall in quality they are strongly suggestive of cavity formation or bronchi dilatation.

A systolic murmur at the base of the heart is frequently present. I the later periods of the disease a mitral murmur of relative insufficien is often present in addition to other evidences of failing compensation.

Diagnosis.—In view of the number of diseases associated wi bronchiectasis it is not surprising that the condition is so frequent overlooked clinically. The following conditions must be considered:

Tuberculosis.—If the bronchiectasis occurs in the lower lobes the should be no confusion as tuberculosis almost invariably starts at t apex and extends downward. If the bronchi of the upper lobe a dilated it is not possible to distinguish the two conditions, inasmuch a tuberculous excavation and the dilated bronchi give rise to the same physical signs. Fig. 221 illustrates very clearly how a mistake might occur. When one entire lung is involved or both lungs are implicated the true nature of the trouble cannot be determined without a sputum examination; and in any case this always should be done. The majority of mistakes are due to neglect of this simple procedure. In examining the sputum it is important to keep in mind that acid-fast streptothrix organisms not infrequently occur in bronchiectasis. To the inexperienced these organisms could be mistaken very readily for tubercle bacilli. In a case under observation at the Phipps Institute, my colleague, Paul Lewis, found that the sputum contained a streptothrix which in the size of the individual organism closely resembled the tubercle bacillus. Instead of being clumped, however, the organisms were arranged in long chains and interlacing filaments. They resisted a 20 per cent. sulphuric acid solution but were readily decolorized with 30 per cent. nitric acid. Guinea-pigs inoculated with the sputum failed to develop tuberculosis. When both tuberculosis and bronchiectasis coexist it is often impossible to recognize the presence of dilated bronchi. They are to be suspected if extreme clubbing of the fingers is present and if in addition, there are signs of a cavity in the area about the angle of the scapula. Tuberculosis alone does not produce extreme clubbing of the fingers. The history of the case will also indicate in some instances, that the bronchiectasis antedates the tuberculous infection.

Moore[1] has studied 25 cases of bronchiectasis by means of *roentgen rays.* He states that a definite diagnosis of the condition was made by this means in 19 cases, a diagnosis of probable bronchiectasis in 2, while in four cases the roentgen diagnosis was incorrect. He distinguishes three types, namely, the infiltrative, the cylindrical and the sacculated. The infiltrative type appears in the roentgenogram as a more or less stringy increase in density along the course of the bronchi, usually localized in the lower lobes and radiating outward from the hilus to the periphery of the lung. This type is not distinctive and is readily mistaken for simple thickening of the bronchial wall.

Abscess of the Lung.—In abscess of the lung the characteristic feature is the sudden expectoration of a large quantity of mucopurulent material, often of a sweetish odor. In the majority of cases of abscess the patient is acutely ill or has recently recovered from an acute illness. In bronchiectasis the development of the disease is gradual and not sudden as in abscess. In the case of a small chronic abscess the distinction is difficult. An X-ray examination may be of service. In case of doubt an exploratory operation should be advised as an abscess, if present, can be cured if proper drainage is secured.

Loculated Empyema.—At times an encysted empyema, especially when situated between the lobes of the lung, ruptures into a bronchus. Under these circumstances it is essentially the same as a pulmonary abscess.

Gangrene of the Lung.—Pulmonary gangrene is usually sudden in onset and may be one of the sequels of a pulmonary infarct, croupous or broncho-pneumonia or tuberculosis. If gangrene of the lung occurs in an individual with a history of long standing bronchitis it may be very difficult to distinguish it from bronchiectasis with gangrenous changes in the bronchi. The presence of elastic tissue in the sputum would be in favor of pulmonary gangrene.

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[1] *Amer. Jour. Roentgenology*, Nov., 1916.

Chronic Bronchitis.—As a rule the presence of dilated bronchi in a case suffering from chronic bronchitis can only be surmised. It should be suspected if there is marked clubbing of the fingers and there is in addition the expectoration of thin, mucopurulent material with a fetid odor. There may also be signs indicative of a cavity especially in the lower lobes of the lungs.

FETID OR PUTRID BRONCHITIS

In those cases in which bronchiectasis, gangrene, or cavities containing a fetid secretion can be excluded and in which there are the physical signs of bronchitis, it has been customary to designate the condition as fetid or putrid bronchitis. The affection is in reality a form of bronchiectasis. In such cases the dilatation of the bronchi is not sufficiently marked to give the physical signs commonly associated with bronchiectasis. Since the introduction of the X-rays, however, it has become apparent that cases which in former times, would have been classed as fetid bronchitis are in reality examples of bronchiectasis. By means of stereoscopic plates small dilatations of the bronchi can be shown. Aside from the physical signs the clinical features of fetid bronchitis are essentially the same as those occurring as the result of well-marked dilatation of the bronchi. The cough may be paroxysmal in character and followed by the expectoration of a foul smelling sputum. The expectorated matter is usually thin, grayish-white in color and on standing separates into three layers: the upper layer is frothy and often of a greenish color, the middle layer is serous in character and slightly cloudy and the third layer consists of a sediment in which may sometimes be found small dirty yellow masses known as Dittrich's plugs. As in the case of true bronchiectasis abscess of the brain is not an uncommon terminal event.

BRONCHIOLECTASIS

This term is applied to a form of bronchiectasis in which the pathological changes are limited to the bronchioles. Although this type of the disease was described many years ago by Carr,[1] of England, it has been almost entirely overlooked in this country.

Etiology.—The condition is not common and occurs almost exclusively in young children who are poorly nourished, syphilitic or rachitic. The exciting cause is generally some one of the acute infections such as whooping cough, measles, diphtheria, etc. Bronchiolectasis is, as a rule, an acute affection.

Morbid Anatomy.—The condition is bilateral and may involve a large part or even all of both lungs. The lungs are usually bulky and scattered over the surface there may be a number of small transparent bladder-like elevations which correspond to the small cavities within the lungs. On section the lungs present a worm-eaten or honeycombed appearance due to the numerous small cavities. The cavities are small, the largest about the size of a pea, have smooth walls and contain either air or a frothy mucus. The bronchi are unaffected. In addition to the dilatation of the bronchioles there is usually present a peribronchial inflammation, patches of broncho-pneumonia and some compensatory emphysema.

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[1] *Practitioner,* 1891, vol. xlvi, p. 87.

Occasionally a chronic form of the disease occurs. In such cases chronic interstitial changes take place in the pulmonary tissue surrounding the dilated bronchioles. This leads to fibrosis of the affected parts with retraction of the lung.

Symptoms.—Cough is the most prominent symptom and as the disease advances tends to become paroxysmal. It may resemble whooping cough. In very young children there may be no expectoration as it is swallowed, but if the child vomits, pus may be mixed with the stomach contents. Fever may be absent but with the successive development of patches of broncho-pneumonia the temperature often rises to 103° or 104°F.

Anemia and emaciation are usually marked features in children who develop this condition.

As a rule the disease is acute and death may occur as early as two weeks from the onset. In some instances it becomes chronic and may exist for three or four years or longer.

Physical Signs.—In the chronic cases marked clubbing of the fingers may be noted. As the disease is bilateral in most instances there is no noticeable difference in the two sides. Scattered râles throughout both lungs may be heard and they may be of all varieties. If they have a ringing metallic quality they are very suggestive of cavities. The percussion note and breath sounds may show little if any abnormality. If the process is more or less localized in one or both lower lobes the percussion note may be impaired or it may have a tympanitic quality. Under these circumstances the breath sounds may be suppressed, broncho-vesicular or even bronchial, depending somewhat on the presence of an associated pneumonic process.

Diagnosis.—*Acute tuberculosis* is the condition most likely to be confused with bronchiolectasis. The physical signs in both conditions may simulate each other closely. Fowler has pointed out that in bronchiolectasis the general symptoms are not nearly so severe as one would expect from the physical signs and that improvement of the child may be noted while the evidences of pulmonary damage are increasing.

In acute tuberculosis fever is nearly always a marked feature, and emaciation is progressive and marked. If sputum cannot be obtained the pus in the vomited matter should be examined for tubercle bacilli.

FOREIGN BODIES IN THE AIR PASSAGES

The aspiration of a foreign body into the air passages is now recognized as a relatively common accident. But while the number of instances reported in the literature is in the aggregate fairly large, the
ity of seeing many of these cases does not often fall to the lot
l observers. The first detailed study of the results of foreign
ng in the air passages was that made by Stokes.[1] In 1854
ributed a monograph on the subject. Since that time there
a number of contributions. In 1909 Eicken[3] reported 303

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[1] "Diseases of the Chest," New Sydenham Society.

[2] "Foreign Bodies in the Air Passages."

[3] *Deut. Klinik*, vol. xii, 1909.

cases in which the bronchoscope had been used to extract a foreign body. The most comprehensive presentation of the subject in American literature has been made by Chevalier Jackson.[1]

Etiology.—A great variety of articles may gain entrance into the air passages. The object may be one normally taken into the mouth, such as an article of food; or it may be one held in the mouth for convenience, such as a pin; or, as in the case of small children, toys or other small objects may be placed in the mouth. The aspiration of the foreign body usually follows some sudden and unexpected inspiratory effort such as may accompany coughing, laughing, a fright or a blow on the back. Under these circumstances the foreign body may enter the larynx during "what may be termed a moment of surprise on the part of the epiglottis while raised in the act of a sudden deep inspiration." The most frequent manner in which a foreign body gets past the protective mechanism and into the air passages is described by Jackson as follows: The intruder is detected escaping downward over the dorsum of the tongue into or toward the pharynx. The tongue and muscles of the fauces and pharynx make a robust effort to catch and extrude the intruder, resulting in gagging, hawking, choking or coughing. These movements are followed by a sudden and deep inspiration. It is this inspiration blast that carries the intruder into the lower air passages, the protective mechanism being held in abeyance by the coördination usual during the inspiratory phase. In other instances, insensibility of the upper air passages as the result of sleep, narcosis or coma may permit the entrance of a foreign body. Occasionally a foreign body, in the form of food, enters the larynx during the act of vomiting.

Of 590 cases in Jackson's series,[2] 492 (81.6 per cent.) were patients under fifteen years of age. This is to be explained by the fact that small children have a habit of placing all sorts of objects in their mouths.

Any article capable of passing the glottis may be drawn into the larynx during a forced inspiration. Among the commoner objects may be mentioned needles, pins of various kinds, tacks, small toys or parts of toys, pieces of meat, seeds of various kinds, especially peanuts and fragments of bones. In one case observed by me an extracted tooth entered the larynx during nitrous oxide anesthesia.

Morbid Anatomy.—Complete blocking of the laryngeal opening by a foreign body is fortunately not common, although such accidents do occur and may be followed by death within a few minutes. In such cases a large piece of meat is the most common offender.

Having passed the glottis the foreign body may lodge in one of the ventricles of the larynx. In the majority of instances the object passes down the trachea and in about 80 per cent. of the cases enters the right bronchus. Almost invariably the foreign body, if it passes the main branches enters the lower lobe branch. There are but four cases recorded in which an upper lobe bronchus was invaded. The great liability of the right side is due to the fact that the right primary bronchus is larger and straighter than the left (see Figs. 225 and 226). The passage downward of the foreign body is facilitated by gravity and by the inspiratory widening of the trachea and main bronchi. The rapidity with which the foreign substance passes downward is due in part to its size and part to the shape of the object. In any case there is produced irritation and spasm of the larynx and trachea and more or less severe paroxysms of coughing in the effort to dislodge the object. As a rule the severity of the symptoms subside with the final lodgment of the foreign body in one of the larger branches of the bronchial tree.

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[1] "Peroral Endoscopy and Laryngeal Surgery," 1915.

[2] *Trans. of Sec. on Laryngology, Otology and Rhinology*, 1917, p. 36.

Spontaneous expulsion of the foreign substance may occur at an early period or months or years may elapse. In one case under my care a tooth was coughed up eighteen months after it had aspirated into the bronchi; in another case a brass-headed tack was expelled four or five years after it had entered the bronchus. In 124 cases recorded by Durham in which recovery followed spontaneous expulsion, the interval in 28 of the cases was from one to seventeen years. Jackson has reported cases in which the foreign body was removed some years after the initial accident; in one case an interval of twenty-six years had elapsed.

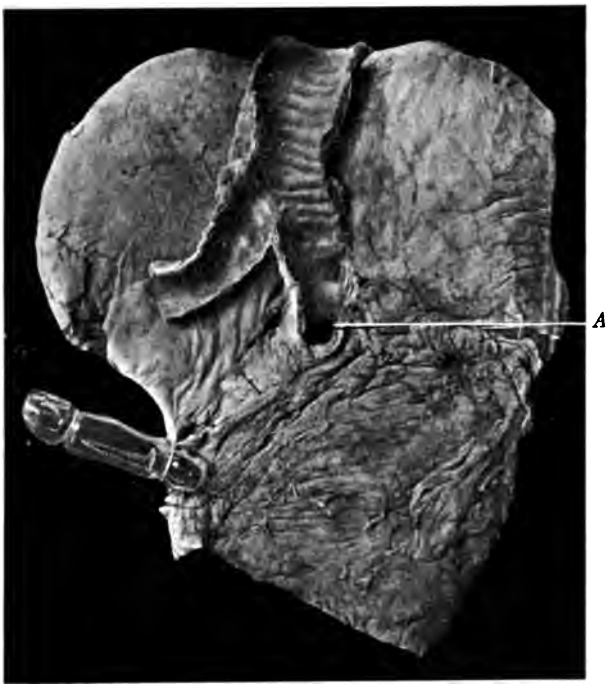

FIG. 225.—Trachea and bronchi viewed from behind. Note the larger size of the right bronchus and its straighter course. The nail shown in the glass tube had lodged in the right bronchus at *A* where it ulcerated through into the lung. (*Jefferson Medical College Museum.*)

Spontaneous expulsion will depend on the nature of the foreign body. If it is small and devoid of sharp points it may be coughed up but if it is long, narrow and sharp-pointed, such as a hair pin or a safety pin, the only hope of relieving the patient lies in surgical interference or the use of the bronchoscope.

If the foreign substance remains in the larynx edema may develop later. The end results of lodgment in one of the bronchi are varied.

The first change is simple inflammatory swelling of the mucous membrane. If the substance remains the inflammation persists, eventually becoming chronic and with it there is apt to be some ulceration. As bacteria are commonly carried by the foreign body infection occurs and this may rupture through the bronchus giving rise to a localized abscess. In addition there is usually more or less of an inflammatory change or abscess formation in the surrounding pulmonary tissue. This eventually becomes chronic and gives rise to fibrosis with or without dilatation of the bronchi in the affected portion. Partly as the result of inflammatory changes which swell the mucous membrane and partly as the result of the foreign body itself the bronchus is obstructed, ending in an abscess below the foreign body. Later sloughing or ulceration follows in the tissues surrounding the foreign body and permitting the slow escape of the pus. The bronchial wall may be destroyed by ulceration and chondrial necrosis. In some cases the foreign body may fall to the bottom of the abscess cavity or as the result of being firmly imbedded it may remain fixed in its original site. Although the bronchial stenosis is usually followed by dilatation of the bronchi below the point of obstruction the air passages may remain normal in size although filled with pus. The term "drowned lung" has been suggested for this condition. Later, if the obstruction is not removed, a true bronchiectasis may develop. In the case of a rounded object complete occlusion of a bronchus may

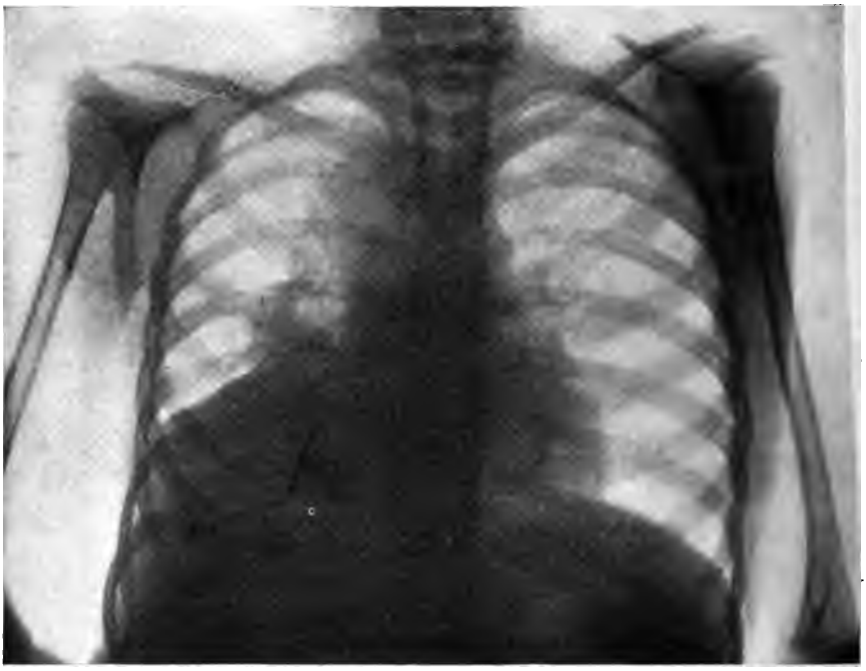

FIG. 226.—No. 14914 (Phipps Institute). Child age six. Cough, 3 years. Physical signs of a chronic inflammatory condition at right base. Onset of symptoms indefinite. X-ray showed fibrosis of lung and dilated bronchi, due to a bent hatpin measuring 2⅝ inches in length with the head down and the point up.

occur with collapse of the lung tissue to which the bronchus is distributed. In some instances a fibrinous or serofibrinous pleurisy occurs. Rarely the visceral layer of the pleura is perforated and an empyma or pneumothorax develops.

Broncho-pneumonia or lobar pneumonia sometimes develops within a few days of the aspiration of a foreign body. If the foreign substance consists of a bolus of food and the particles enter the finer bronchi a septic bronchopneumonia is almost certain to occur.

Symptoms.—The symptoms produced by the aspiration of a foreign body into the air passages may be divided into primary and secondary manifestations, between which a period of comparative latency may intervene.

Immediately following the entrance of the foreign body into the larynx the patient is seized with a paroxysm of coughing, becomes dyspnoic and cyanosed. In most instances the patient experiences the sensation of suffocation. If the body is large the patient may present the appearance of one strangling to death. The eyes protrude, the face becomes livid, and there may be vomiting, convulsions and finally loss of consciousness. Death may occur within a few minutes—but fortunately this accident is relatively rare. The violence of the initial symptoms may last from a few minutes to an hour or more and may recur at intervals.

Succeeding the acute symptoms there may be some hoarseness if the vocal cords have been injured or the foreign body remains in the larynx. There may be a sense of soreness beneath the sternum and very often the tenderness is referred to the right of the sternum. Expectoration, if present, is scanty and may be blood-tinged. Dyspnea and cough may persist, recur in paroxysms or entirely disappear for a time.

Price[1] in reporting 30 cases from Chevalier Jackson's clinic states that the immediate effect produced by the foreign body was choking spells in 21; coughing in 14; dyspnea in 14; dyspnea occurring later in 4, cyanosis in 5, spasmodic cough in 4, bloody expectoration in 5, inspiratory crowing in 2 and aphonia in 1. Jackson emphasizes the importance of these initial symptoms. In a number of cases observed by him the possibility of a foreign body being present had been ridiculed or ignored. It must not be forgotten that a foreign body may lodge in the bronchi without causing symptoms, even cough, for an interval of a number of weeks between the initial choking and the vague onset of bronchial and later, general symptoms. Thornval[2] has reported 16 cases some of which had no symptoms whatever until some time had elapsed. All but 2 of these cases were children.

The secondary manifestations develop insidiously. The cough gradually becomes worse and is attended with considerable mucopurulent expectoration. In some cases tuberculosis is suspected because of the hectic type of fever, sweating and loss of weight. In others there is a gradually developing fibrosis of the affected portion of the lung, dilatation of the bronchi and all the symptoms of bronchiectasis, namely: a paroxysmal cough, large quantities of purulent sputum, some fever and clubbing of the fingers. The sudden expectoration of a large quantity of purulent material may be the first intimation of a pulmonary abscess. Exertion or change of posture sometimes brings on a paroxysm of coughing.

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[1] *Penna. Med. Jour.*, December, 1915.

[2] *Ugeskrift for Laeger*, Dec. 23, 1915

No matter what the particular lesion is, permanent lodgment of a foreign body in the lung is usually attended with cough, the expectoration of purulent sputum, some fever and often with loss of weight.

If the patient is seen within a day or so of the accident the upper air tract should be examined with a laryngoscope. If there is available one who can use the bronchoscope this method should also be utilized. In all cases stereoscopic X-ray plates of the chest should be made.

Physical Signs.—As a rule there is nothing distinctive in the physical signs which may be encountered in cases with a foreign body in the lungs. The signs are usually those of some other familiar condition such as fibrosis of the lung, bronchiectasis, abscess, empyema, or pneumothorax.

Jackson[1] has described a new sign, the asthmatoid wheeze, which he believes will be of great value, especially in those instances in which the foreign body is permeable to the X-rays. The asthmatoid wheeze is elicited by oral auscultation, the ear of the examiner being placed close to the open mouth of the patient. It resembles the wheezing of the asthmatic patient, but is drier. It is best elicited during forced and prolonged expiration or following coughing. In many cases it is readily detected during mirror laryngoscopy in older children and adults.

Diagnosis.—In the absence of any history of the accident one should always be alert to the possibility of a foreign body in cases simulating *tuberculosis* but in which the physical signs are limited to one of the lower lobes. The same is true of bronchiectasis. In the presence of these two conditions a foreign body is constantly overlooked.

In some instances the foreign body may completely block one of the large bronchi and cause collapse of all or a large portion of a lower lobe. In such cases the absence of breath sounds and dulness may lead to a diagnosis of fluid.

Inasmuch as a variety of conditions may be caused by the lodgment of a foreign substance in the bronchi or pulmonary tissue the physical signs give no direct clue as to its presence. The occurrence of such a possibility, however, is always to be borne in mind when the signs are limited to the lower lobes, especially the right lower lobe. We have made it a routine practice at the Phipps Institute to have an X-ray examination made in all cases of this nature and every now and then are rewarded with the finding of a foreign body. Even if the X-rays fail to show a foreign body, valuable evidence is often obtained by the showing of a localized inflammatory area in the lung which may suggest the true nature of the trouble. As the value of the X-rays is limited chiefly to metallic objects the bronchoscope should be employed whenever possible. By using the latter instrument, pieces of bone, beans or other articles of food may be discovered which will often escape detection in the X-ray plate. In the hands of a skilled operator bronchoscopy is the method of choice, as it is possible to directly locate the foreign body and at the same time remove it. Price warns against the use of the bronchoscope by those not possessing the requisite skill. He reports three cases in which broncho-pneumonia followed the trauma produced by unsuccessful attempts to use the instrument.

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[1] *Amer. Jour. Med. Sc.*, Nov., 1918.

CHAPTER XXII

DISEASES OF THE LUNGS

TUBERCULOSIS OF THE LUNGS

"Consumption of the lungs may be traced with certainty in the writings of every period as far back as the earliest attempts of the ancient world to deal with medicine according to method. History does not inform us, however, of the extent to which the malady had been prevalent during former times in various parts of the world . . . . But there can be no question that pulmonary consumption has held at all times and among all civilized peoples a foremost place among the national diseases. In our own age, at all events, it occupies one of the leading positions in the statistics of mortality."[1]

The above quotation holds as true to-day as when written forty years ago and in spite of the fact that the mortality rate of this disease has been steadily falling, tuberculosis must still be looked upon as one of the great scourges of civilization. In this country it is the cause of about one-tenth of the deaths annually.

So far as diseases of the chest are concerned a thorough knowledge of the various manifestations of tuberculosis is essential. The term "protean" is often applied to other diseases but in no instance is the designation more applicable than in the case of tuberculosis. *In every individual who presents himself with symptoms referable to the thorax the possibility of the trouble being due to tuberculosis must always be considered, no matter whether the affection is acute or chronic.*

Not only is tuberculosis the most important single disease the practicing physician has to deal with but in addition it possesses the greatest interest to the sanitarian by reason of its widespread prevalence among domestic animals. Tuberculosis is not uncommonly found in pigs, and among cattle it is very prevalent. As we derive all of our milk and a large proportion of our meat from bovine sources, it is essential that measures be taken to prevent the spread of the disease by the adequate inspection of abattoirs and dairy herds and further, by the pasteurization of all milk.

Three types of tuberculosis of the lungs are recognized: (1) chronic pulmonary tuberculosis; (2) acute pulmonary tuberculosis; and (3) fibroid tuberculosis.

CHRONIC TUBERCULOSIS OF THE LUNGS

Chronic pulmonary tuberculosis is also known as tuberculosis of the
onic ulcerative tuberculosis, phthisis and consumption.
.—The cause of tuberculosis is the tubercle bacillus, the source
in the vast majority of cases, the sputum from human cases
esser extent, the milk from tuberculous cows.
rcle bacillus gains entrance into the body in a number of ways:

"Handbook of Geographical and Historical Pathology," 1876.

(1) The most important mode of infection is by *inhalation*. In the great majority of instances the infection occurs from the inhalation of dust in which are carried the tubercle bacilli. Careless habits on the part of a consumptive are entirely responsible for this. The sputum, which is recognized as the principal source of infection, is not dangerous in the moist state and if, in every instance, it was destroyed in this condition, the disease practically could be stamped out. Spitting on the floor in the house or the immediate surroundings of the home or in working places permits of the sputum becoming dried and mixed with the dust. Houses or working places which are dark and badly ventilated are especially favorable to prolonging the life of the tubercle bacillus which quickly succumbs when exposed to the light and air.

Flügge advanced the theory that the chief source of infection was through the minute droplets which are expelled during coughing. These he believed are inhaled. While they may, in some instances, play a part, it is now generally accepted that the inhalation of infected dust is the chief source of the disease in adults.

In most instances exposure to the disease must be prolonged and under the conditions mentioned above. There is no danger from the expired breath of consumptives nor from associating with those who are careful in their habits. For this reason a tuberculosis sanatorium is probably the safest place one can be so far as the danger of infection is concerned. Statistics dealing with those who have been employed for years in tuberculosis sanatoria show that infection practically never occurs among these workers.

Until the study of *marital infection* by Pope and Pearson[1] appeared it was generally believed that the infection of a wife by her husband or *vice versa*, was very common. These observers from an analysis of a large number of cases, concluded that while there was some slight danger from this source the majority of such instances could be ascribed to infection from other sources, to the presence of the necessary diathesis and to assortive mating.

2. For some years there was a tendency to minimize the danger from inhalation and place *infection by ingestion* in the first place. Infection through the digestive tract undoubtedly plays an important part but not to the extent once believed. The point of entrance may be through the tonsils or the intestinal mucous membrane. The tonsillar and intestinal routes are more common in childhood than adult life. In young children the intestinal mucous membrane seems to be especially permeable and many children are known to become infected by being fed milk containing bovine tubercle bacilli.

3. *Localized infection* may occur in those who perform autopsies, in butchers, in handlers of hides, or in those who handle clothing or utensils used by consumptives. As a rule the infection takes the form of a small, localized, reddened, granulation mass on the fingers or hand. Occasionally the infection may not appear at the point of inoculation but is carried to the group of lymph nodes in the axilla.

4. The rarest mode of infection is the direct transmission of the disease from the *mother to the fetus*. This is so unusual as to be negligible. To date only fifty-one cases of undoubted congenital tuberculosis are on record. An investigation in Copenhagen showed that of 317

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[1] "Marital Infection," Draper's Company Research Memoirs, 1908.

born infants not one was tuberculous. Occasionally, however, the ease will show itself comparatively shortly after birth as the result intense exposure. There has recently been reported two prematurely n infants both suckled by their consumptive mothers. One developed ns of pulmonary tuberculosis a month after birth, the other three nths later.

Theobald Smith has pointed out that though a strictly bacterial ease and introduced into the body by the tubercle bacillus, which is ays derived from some preëxisting case of the disease, tuberculosis fers, nevertheless, from most diseases in many important particulars. s unknown beginnings in the body and its insidious march after it has ce gained a foothold are responsible for the existence of a large number cases in all stages of the disease. In the earlier stages, while the disease is still restricted to a single focus, the individual is to all outward pearances in perfect health. The disease may remain in this quiescent age indefinitely and then for one reason or another become active. If e generally accredited theory be accepted, namely, that an initial nplantation takes place early in life in the majority of people, what is that determines that one individual will later in life become tuberculous hile another escapes? The following factors must be taken into ccount:

1. We have to consider the question of *resistance*. Were it not for e fact that the great majority of people possess an immunity to tubercu- sis the death rate would far exceed what it is at present. The degree f immunity possessed by the particular individual varies greatly. In any it is probably absolute; in many more the susceptibility to the fection is very marked and they readily fall victims to the disease. he great majority of people, in all probability, possess sufficient resis- ance to the growth of the tubercle bacillus providing this resistance s not lowered by other causes.

Another factor which must be considered is the *degree of virulence* f the tubercle bacillus. It is well known that different strains of the rganism vary tremendously in virulence; some possess very little power r mischief while others are capable of producing very destructive sions. While we lack sufficient proof as to the effect of a strain of nown virulence, it is not unreasonable to assume that when the disease rogresses but slowly and extends over a period of years, the resistance f the individual is marked and that in addition the virulence of this articular strain is not great. On the other hand rapidly progressing ses probably represent instances in which the resistance is slight and he virulence of the organism is marked. As I have pointed out in the iscussion of the pathology of the disease resistance to the tubercle acillus is expressed in the capacity of the lung tissue to form a fibrous all about the lesion. Thus in those with good resistance the progress f the disease is entirely arrested or greatly retarded by the formation f fibrous tissue. This is often well seen in the structure of the individual bercle about which a fibrous wall is built. In the acute cases, those bviously of low resistance, the absence of fibrous tissue is a striking ature of the lesions.

In addition to the resistance of the individual and the virulence of e tubercle bacilli Cobbett[1] believes, and I think rightly, that the

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[1] "The Causes of Tuberculosis," 1917.

dose has an important bearing. Resistance, he believes, varies in different individuals and in the same individual at different times. A dose fatal to one may be innocuous to another. The dose is often too small to infect anyone; at other times so large that it kills in spite of resistance. Large doses are to be looked for in those in close proximity to a tuberculous patient whose cough is spraying tubercle bacilli into the air constantly or whose carelessness in spitting fills the air with those organisms or as the result of a draught of milk from a cow with a tuberculous udder.

That the *white races* have acquired a certain degree of immunity during the centuries to which they have been exposed to the disease is consistent with what we know of other infectious diseases. Two races, which in their primitive state were free from tuberculosis, have paid a heavy toll through association with the white race. The *North American Indian* has been especially susceptible, tuberculosis being one of the most potent causes of the rapid decimation of this race. For the year 1915 the greatest single cause of death among the Indians was tuberculosis which was responsible for 35.08 per cent. of all deaths. The *Negro* is another example of a race which has been exposed to the disease but a short period and in which sufficient time has not elapsed to establish much immunity. Not only is the negro extremely susceptible but, as a rule, the disease pursues a much more acute course than in the white man. The death rate from tuberculosis among negroes is from three and a half to four times that of the whites.

A recent report by Marrable[1] illustrates the virulence of tuberculosis when introduced on virgin soil. He states that up to 1900 tuberculosis was unknown in Central Persia. Introduced into a village, from an outside source, 100 cases developed between 1900 and 1908. Two facts stand out: first, the rapidity of the disease; rarely did it last more than six months, never more than a year, secondly, its mortality, all died.

2. *Heredity.*—Until comparatively recently it was taught that tuberculosis was an inherited disease. The basis for this belief lay in the fact that it was a common observation that the children of tuberculous parents very frequently developed the disease. We now know that direct inheritance of the disease is extremely rare, so much so as to be practically negligible. The real cause of tuberculosis being a family affection is due to its transmission from the infected parent to the child after birth. This may occur as the result of the careless disposal of the sputum in the home; by kissing; or by infection of the child's food. Owing to the more intimate contact between the mother and the child it is well recognized that a tuberculous mother is more dangerous to her children than a tuberculous father.

Although a child is rarely born tuberculous, there is reason to believe that, in some instances, the children of tuberculous parents are more susceptible than others and that when exposed to infection they offer little resistance. Karl Pearson, especially, has been insistent on the importance of a tuberculous diathesis.

3. *Reinfection.*—As already stated many authorities place the time of infection during the early years of life, the belief being that the bacilli lie dormant until some time later when, for one reason or another, the individual's resistance becomes lowered and the disease becomes active.

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[1] *Dublin Jour. of Med. Sc.*, July, 1917.

An individual may, however, become reinfected in adult life and succumb to this second implantation of the tubercle bacilli. Paul Lewis[1] is of the belief that the primary infection in early life has little influence on the disease as it presents itself in adults. This judgment is based on his experience with bovine vaccination where the immunity seems not to persist much longer than do living bacilli introduced as a preventive. Applied to the human type of the disease this fact may be interpreted as follows: There is a high incidence of infection in early childhood, very possibly owing to an especial permeability of the intestinal mucous membranes in that period. By the adolescent period, however, most of the infected children are either cured of the disease or have died of it. Following the twentieth year a fresh period of high incidence occurs, probably owing in part to the failing natural immunity of the lungs and in part to increased exposure. There can be no doubt that infection in adult life is far more frequent than is ordinarily taught. This is apparent from the fact that healthy adults, with no history of tuberculosis in the family, not infrequently acquire the disease by moving into a house infected by a former tenant.

4. *Insanitary Surroundings.*—For years it has been recognized that tuberculosis is essentially a house disease. It is in the home, in the vast majority of instances, that the disease is acquired and here from its inception to its close the entire drama is played. The worse the living conditions are, the higher will be the incidence of tuberculosis, and this applies to the isolated country house as well as the congested city slum. In considering the influence of the home surroundings, two factors must be taken into account: first, the home itself; and secondly, the habits of the people.

Much emphasis has been placed on the badly ventilated and lighted house as a predisposing cause of the disease, and of this there can be no doubt. Another factor, and one which is not so well recognized, is the unhygienic habits of the people themselves. In a study made by the Phipps Institute[2] it was found that many houses which had little to commend them were inhabited by people who were cleanly in their habits; on the other hand, model houses were often found to be tenanted by people who made no pretense of observing the simplest rules of hygiene. Perhaps the greatest lesson which the crusade against tuberculosis has taught is the importance of hygiene. Not only must better houses be provided but in addition the people themselves must be impressed with the dangers of insanitary habits. They must be taught that irregular meals, food of poor quality, insufficient sleep and dissipation in any form lead to a lowering of the resistance and thus render the individual more susceptible to infection. In an occupational study made at the Phipps Institute[3] it was shown that among those workers who took their meals irregularly, and among those who did not get sufficient sleep the incidence of tuberculosis was much higher than in the case of those leading a regular life.

In regard to the influence of insanitary dwellings the experience of Liverpool is instructive. The municipal government tore down large sections of defective dwellings and erected in their place sanitary houses.

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[1] *Journal of the Outdoor Life*, February, 1916.
[2] Eleventh Report, 1915.
[3] Eighth Report, 1915.

The same people were housed in the new dwellings who had occupied the old ones. In this reconstructed district the death rate from tuberculosis fell from 4 per 1000 to 1.9 per 1000. A very striking example of the effect of destroying a slum district was noted after the great fire in San Francisco. Prior to the fire in 1905 the death rate from tuberculosis in that city was 274 per 100,000; two years later it had fallen to 179 per 100,000. In 1912 it had fallen still farther to 153 per 100,000.

Poverty and tuberculosis go hand in hand. This is due to the fact that lack of money forces the very poor into the most insanitary districts and in addition does not permit of their obtaining suitable food. Both of these factors lower the resistance and make the individual a ready victim of the disease. The effects of privation and food, insufficient in quality or quantity, has been shown strikingly in the devastated regions of France and Belgium during the recent war. Indeed the effects have been felt also in the neutral European countries and in uninvaded portions of the warring countries. Reports from nearly all the European countries during the past three years all tend to show that tuberculosis is on the increase.

5. *Occupation.*—The influence of occupation as a predisposing cause of tuberculosis has been emphasized over and over again. There are many employments which are credited with being bad, but which in reality are not, much of the evil that is associated with them being the result of contributory factors which are not directly connected with the work itself. It is well to remember that in the badly ventilated state of one factory compared with another, the home life and surroundings of the work people, poverty, heredity, age and sex are to be found conditions that favor the production of ill health, and are therefore, not to be ignored. Usually it is a gradual deterioration of health that is produced. It cannot be emphasized too strongly that *it is impossible to determine the effect of a given occupation as a predisposing cause of tuberculosis without a knowledge of where and how the worker lives.*

6. *Acute and Chronic Infections.*—As the result of some acute or chronic disease the individual may be rendered more susceptible to reinfection with tuberculosis, or as the result of lowered resistance, a quiescent tuberculous lesion may become active. *Acute catarrhal conditions* of the upper respiratory tract, measles, whooping cough, etc., are not infrequently followed by tuberculosis.

The importance of damp soil and damp houses was emphasized many years ago by Henry I. Bowditch, who showed that such conditions were productive of catarrhal affections of the respiratory tract and that they in turn predisposed to tuberculosis. Sims Woodhead also has pointed out that in pulmonary tuberculosis the most important predisposing cause appears to be a catarrh. This is most apt to occur in portions of the lung where expansion and contraction are weak or imperfect, as at the apices.

Among the *chronic diseases* in which tuberculosis is often a termina infection may be mentioned diabetes, arterio-sclerosis, thoracic aneurism and cirrhosis of the liver.

7. *Pregnancy and the Puerperal Period.*—Many women first manifes evidences of pulmonary tuberculosis during or immediately after th termination of pregnancy. That the incidence of pregnancy and tuberculosis is a frequent one is shown by the figures given by Bacon. In

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[1] OLIVER'S "Dangerous Trades."

913 he estimated that 32,000 tuberculous women became pregnant nnually in the United States, and added that between 44,000 and 8,000 women of child-bearing age die of tuberculosis every year. When he disease becomes active during pregnancy it is probably due to the ncreased demands made on the woman's vitality. When it first shows itself during the puerperal period or undergoes an acute exacerbation at this time, it is due, in large measure, to the violent efforts attending the delivery of the child. It is noteworthy that among women a very large number date the onset of their pulmonary trouble from a pregnancy. In a study of tuberculous pregnant women made at the Phipps Institute by C. C. Norris and myself[1] we found that about 20 per cent. of mild, quiescent cases and 70 per cent. of more advanced cases exhibit exacerbations during pregnancy or the puerperium.

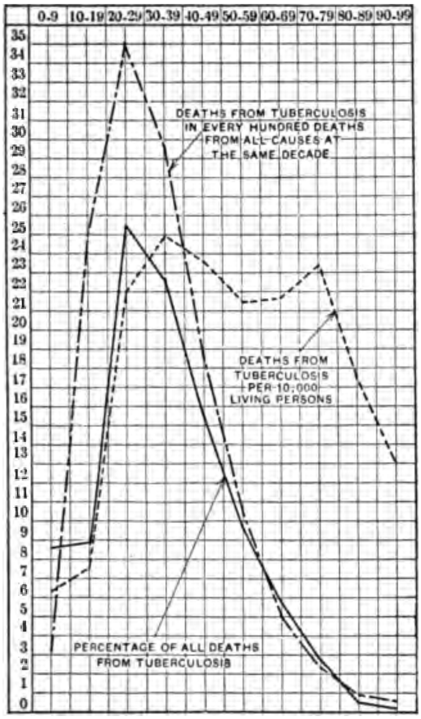

Fig. 227.—Tuberculosis: all forms. Registration area of the United States. The dotted line represents the number of deaths for the disease specified for different decades per 10,000 persons living at corresponding decades. The solid line represents the ratio of deaths from a particular disease at different decades to the whole number of deaths at all ages from the same disease. The broken line in Chart represents the ratio of deaths from tuberculosis at any decade to the total number of deaths from all causes at the corresponding decade. (*Montgomery Amer. Jour. Med. Sc.*, Sept., 1915.)

A consideration of the above etiological factors makes it apparent that over and above the implantation of the tubercle bacillus in the body something additional is needed, in the vast majority of cases, to cause the individual to develop clinical tuberculosis.

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[1] *Jour. Amer. Med. Assoc.*, Feb. 9, 1918.

For some years past I have been able to fix on the determining factor in the great majority of private patients. In one it has been excessive mental anxiety over business affairs; in another the nursing of a relative in a long and tedious illness; in still another, a period of overwork and lack of sufficient sleep. Among the lower class the prolonged exposure to insanitary surroundings and poor food are to be looked upon as the most potent causes.

As already pointed out, no race is exempt from the disease and it is to be met under all sorts of climatic conditions. Tuberculosis occurs at all age periods but the *chronic ulcerative type affecting the lungs is essentially a disease of adult life.* Statistics show that the highest incidence of pulmonary disease is between the twentieth and fortieth years. Montgomery,[1] however, has called attention to the fact that if the death rate is computed for the different age periods in relation to those living, the disease is as prevalent among those of advanced years as in early adult life.

Fig. 227 pictures the mortality curve for all forms of tuberculosis in the registration area of the United States, and shows what has long been known, but is frequently overlooked, that tuberculosis continues unabated its extensive ravages even among elderly perons, though the absolute number of deaths from tuberculosis is actually diminishing. Among those who have emphasized the high mortality from tuberculosis among the aged are Wilson Fox and Cornet. A pamphlet issued by the Maryland State Department of Health entitled "A Brief Review of the Tuberculosis Campaign, 1904–1914," shows that "in the white population beginning with the twentieth year of life all persons are equally liable to death from tuberculosis."

Morbid Anatomy.—A knowledge of the pathology of tuberculosis as it affects the lungs, is absolutely essential in order to gain a clear conception of its various manifestations.

In both its pathological and clinical aspects the disease may exhibit the widest extremes. It may, on the one hand, run an acute course, the duration of which is measured in weeks or, on the other hand, it may pursue a chronic latent course lasting for years. The very acute and the very chronic cases, however, form but a small proportion of the total number. The overwhelming majority of cases of pulmonary tuberculosis is comprised of the type referred to as chronic ulcerative tuberculosis; more commonly known as phthisis or consumption. In this type of the disease the course is rarely continuous but is almost invariably characterized by periods of activity which are succeeded by periods of quiescence or indeed complete arrest of the disease, which may last for twenty years or even longer. The too commonly accepted belief that pulmonary tuberculosis has an average duration of from two and a half to three years, is no longer tenable. In the vast majority of cases the symptoms which seem to indicate the beginning of the trouble, are in reality antedated by years by some symptom or group of symptoms which either have been ignored or have been attributed to some other disease. This may be illustrated by the following case which, while extreme, is by no means uncommon.

A banker, aged fifty-two, came under observation for the first time with a laryngeal tuberculosis and physical signs which clearly indicated an infiltrating process involving the upper third of the left upper lobe. He stated that he had been perfectly well up to three months previously. His history, however, showed that he had had at the age of twenty-two years several pulmonary hemorrhages for which he was confined to bed for a few days. Ten years later he had a severe attack of pleurisy which laid him up for a week. This was followed by no ill effects. Here then we have a case in which the disease gave warnings of its presence thirty and twenty years prior to his becoming definitely tuberculous. During this long period his health was good and he finally succumbed to the infection because his resistance became lowered through overwork and financial worries during a business panic. This man made a complete recovery and is at the present time perfectly well (seven years later).

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[1] *Amer. Jour. Med. Sc.*, September, 1915.

A knowledge of the fact that this type of tuberculosis is subject to the occurrence of periods of slight activity which subside spontaneously, is of considerable practical importance as it indicates that the disease is more or less latent and chronic and that the patient's resistance is good. In this type of case the prospect of recovery is, as a rule, a great deal more promising than in that with an acute onset and no antecedent history of trouble.

The acuteness or chronicity of every tuberculous infection is to a great extent determined by two factors. On the one hand, we have the invading tubercle bacilli striving to multiply and spread and destroy tissue. Opposed to this invasion are the resources of the host which strive to hold in check the invaders. While some of the tubercle bacilli may be destroyed by the phagocytes, the chief defense against them lies in the formation of fibrous tissue which surrounds the tubercles and completely or partially arrests the process. That this is true is evident from the fact that the acute types of the disease show little or no evidence of fibrous tissue about the lesions, while in the more chronic forms of the disease an extensive overgrowth of fibrous tissue constitutes the chief characteristic of the pulmonary lesions. In chronic ulcerative phthisis, therefore, the rapidity or slowness of the progress of the disease is determined, largely, by the capacity of the lung to undergo fibroid changes.

Sims Woodhead has described the initial changes as follows: The first stage is an apical catarrh; that is, a congestion of the blood-vessels of the mucous membrane, accompanied by a slight proliferation of the epithelial cells lining the air cells with an increased pouring out of fluid and an emigration of a large number of white cells. This catarrhal change is noted in portions of the lung where expansion and contraction are weak or imperfect, as at the apices in adults or about the hilus in children.

This breeding ground for the tubercle bacillus may gradually increase in size. If, however, the tissues are well nourished, fibrous tissue is formed. Building up the resistance of the patient by proper treatment aids in bringing this about. If this is accomplished, the bacilli become inactive and the degenerative tissue is partly absorbed and partly transformed into scar tissue. If, however, the process does not become arrested at this point, the disease spreads beyond its original focus and a considerable area of tuberculous consolidation may be the result. The degenerated or caseous material may then become softened and it is during this stage of softening that secondary infection goes on perhaps most readily.

The initial deposit as seen at the autopsy table in individuals dyin from other causes may consist of a single small peribronchial focus (se Fig. 228) or it may consist of several areas the size of a hazel nut. Suc foci represent the coalescence of a small group of tubercles. From thes areas the disease spreads or healing may take place (see Fig. 229).

In those instances in which the disease gains the upper hand th tubercles which at first are discrete, coalesce and in addition spread beyond their original boundaries. Thus at the apex where the origi deposit occurred the infiltration becomes more and more dense a furthermore the tubercles spread downward until the entire lun involved although in the lower portions the infiltration is usually wid scattered.

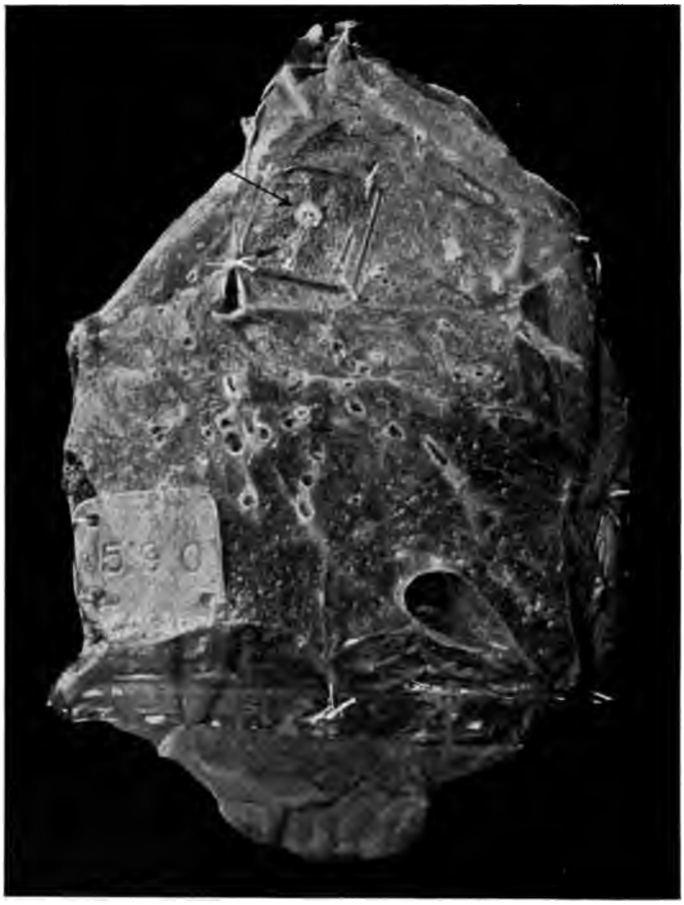


Fig. 228.—Solitary peribronchial tubercle indicated by arrow. Rest of lung nor (*Jefferson Medical College Museum.*)

This constitutes the second or moderately advanced stage. Fi 230 shows this very well. It will be noted that at the extreme apex th infiltration, while very dense, has not replaced entirely all of the pulmo ary tissue so that some vesicular element will be retained in the respiratory sounds. The infiltration becomes less and less marked as the base of the lung is approached. The lower third is apparently free from disease.

The chief characteristic of chronic ulcerative tuberculosis is the formation of cavities. As a rule a cavity is an indication that the disease has progressed to the third or advanced stage of the disease. This is not invariably true, however. The original focus may break down and empty out through a bronchus and the disease become arrested at this point; and the same is true of the second or moderately advanced stage.

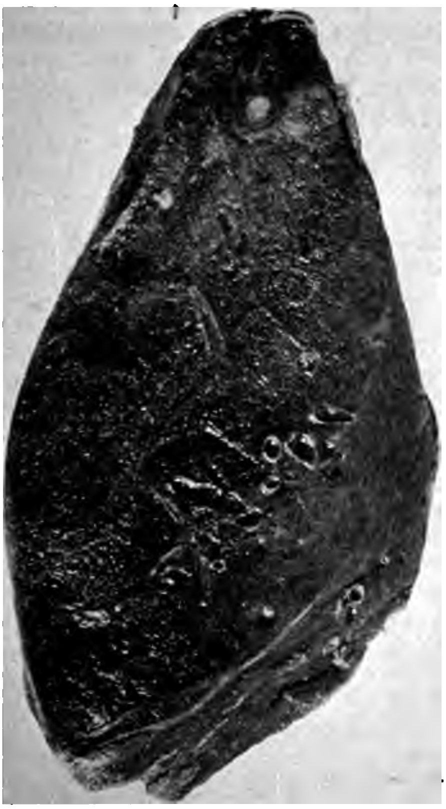


FIG. 229.—Below the apex are seen several calcareous nodules surrounded by fibrous tissue. These represent small deposits of tubercles which have become walled off. It is in this way that tuberculosis is often healed.

In the advanced stages of the disease it is almost the rule to find a cavity at one or both apices and in addition smaller cavities may be present in other portions of the lungs.

The excavation may be represented by a single large cavity with a well-defined wall or it may present a honeycombed appearance in which the cavities are small and communicating with ragged, necrotic walls.

The former type is characteristic of the chronic type of the disease, latter of the more acute forms (see Acute Pulmonary Tuberculosi

It is the present belief that the formation of a cavity is due in part, to the presence of secondary infections with the various pus-pro ing organisms. These organisms produce a liquefying necrosis of caseous areas and thus hasten their breaking down. The final sta the formation of a cavity is the rupture of the caseous and necrotic into a bronchus. As a rule there is but one such communication;

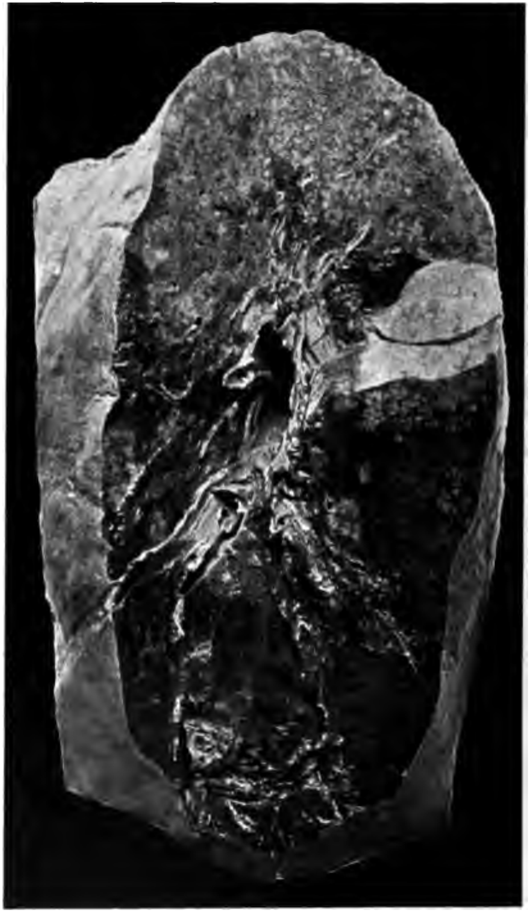


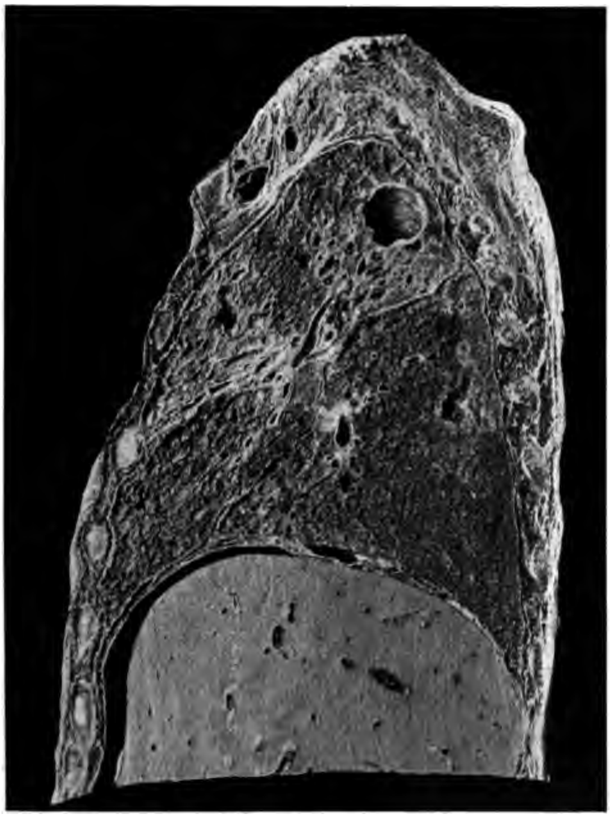
FIG. 230.—Dense infiltration of upper lobe. Areas of conglomerate tubercles in u part of lower lobe.

there may be two or three, in which case the physical signs are apt to indefinite and imperfect.

The longer the tuberculous process lasts the more certainly is exca tion apt to occur although this is not an invariable rule, particularl the chronic fibroid cases. Recently formed cavities have an irreg outline, are ill-defined, the interior is ragged and there is an absence

t wall. When the process is not acute or has remained localized
ne time, a more or less well-formed wall of fibrous tissue develops.
nger the cavity has existed the thicker the fibrous tissue about it.
rmore, the caseous lining of the cavity gradually changes into
ation tissue and this in turn may be replaced by smooth fibrous

. 231 represents a chronic type of infection. The cavity is sharply
d and from its shape seems to have been formed by several smaller
tions which have united. Below the cavity is a scattered infiltra-
ith some fibroid changes and dilatation of the bronchi.



231.—Cavity in apex of right upper lobe and a smaller one anteriorly just
the clavicle. In addition to cavities there is healthy pulmonary tissue and con-
e fibrosis. Illustrates a chronic and slowly progressive type of disease.

. 232 illustrates how extensive the pulmonary excavation may
e. In this instance practically the entire lung has been reduced
brous shell with only a fringe of pulmonary tissue at the base.
view of the extension ulceration which may occur it is remarkable
erforation of the chest wall so rarely occurs. Several such cases
een reported. In one case seen at the Phipps Institute perforation
chest wall was prevented by a rib, which, however, had become

markedly necrosed. In another case seen in the White Have
torium the perforation (a half-inch in diameter) had extended t
the internal intercostal muscle. The external intercostal was ul
covering this was the pectoralis minor muscle and the skin.
case there was during each inspection a distinct bulging about t
of an English walnut and strongly resembled a hernia of the lung

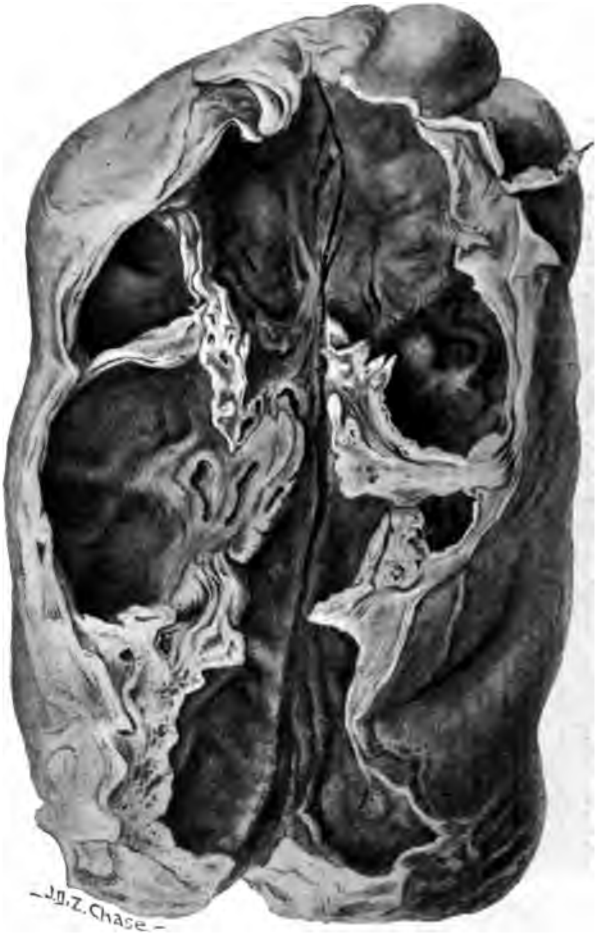


FIG. 232.—Case No. 2388 (Phipps Institute). Upper three-fourths of lung tak
three cavities with great outgrowth of fibrous tissue.

Another important feature of cavities is that those of any consi
size contain in their walls or the trabecula traversing them, blood-
(Fig. 233). As a rule the blood-vessels in and about the cavity
thrombosed and obliterated. If they remain patulous, small ane
dilatations occur in their walls and the rupture of one of these w
rise to a hemorrhage. Fig. 234 represents a cavity filled with blo
in which this accident occurred. (See also Fig. 59.)

n addition to a knowledge of how the disease develops it is important
cognize the routes it follows from the time the initial deposit of
rcles occurs to the final stages. The proportion of cases in which
pex is the primary site of the disease is so large that, generally speak-
the exceptions can be disregarded. Laennec's brief description of
successive changes is worth quoting: "We very often find in the
e lung evident proofs of two or three successive eruptions, and we
then nearly always make out that the primary eruption at the apex
he lung has already become excavated; that the second, situated
nd the first, but a little lower, is formed by tubercles which, in
t instances, are already yellow, though still small; that the third,
ned by gray miliary tubercles, with some yellow points in the center,
upy a still lower zone."

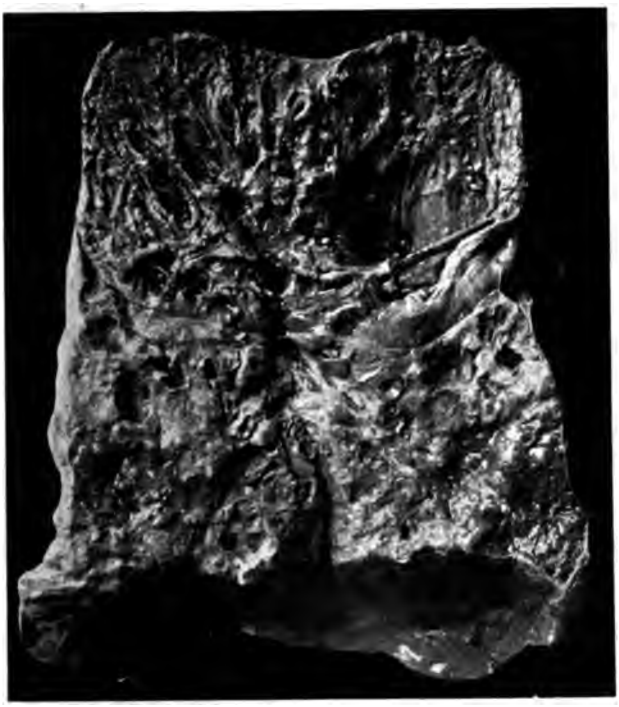


FIG. 233.—Large cavity in upper lobe with blood-vessels laid bare.

These features, namely, the primary deposit in one or the other apex;
extension downward and the presence of the greatest damage where
disease first began, are characteristic of tuberculosis and serve as
uable aids, inasmuch as the physical signs will be more marked at
summit of the lung and while continuous, diminish as the base is
roached. This distribution of the disease also serves to distinguish
erculosis from certain chronic inflammatory diseases of the lung, which
de the base rather than the apex. While these generalizations are
absolute, "the line of march" in the great majority of cases un-
btedly shows a great similarity.

Not only are the apices usually the original site of the disease, but Fowler has shown, a fairly definite area in the apex is the comm starting point. This point is about 1 inch to 1½ inches below the ap and corresponds anteriorly to the supraclavicular fossa or the middle the clavicle. Extending from this point the disease tends to spre backward. As the process spreads, it progresses downward, the in tration about the original focus increasing and gradually thinning ou the base is reached.

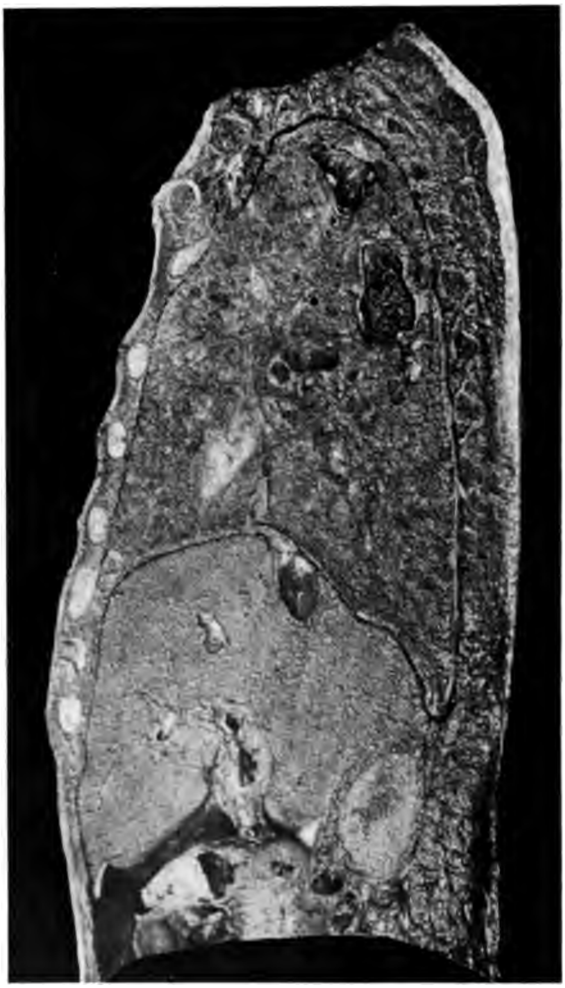


Fig. 234.—Right lung. Advanced tuberculosis. Several cavities, the largest, at bottom of the upper lobe posteriorly is filled with blood clot. (Patient died of hemopty Extensive infiltration and fibrosis of both lobes.

A less usual site for the primary infection occurs at a point wl corresponds to the first and second interspaces below the outer third the clavicle.

Of almost equal importance to the primary infections in the apex re the secondary deposits in the lower lobes. Fowler points out that he involvement of the lower lobe may be very early in the disease and hat although it follows the primary apical infection, may give more definite physical signs because of the doubtful character of the apical findings. As a rule the apex of the lower lobe is first involved. This corresponds to a point opposite the fifth dorsal vertebra in the interscapular region (Fig. 235). From this point the disease tends to spread downward along the interlobar septum, which is roughly indicated by the inner border of the scapula, when the hand is placed on the opposite shoulder.

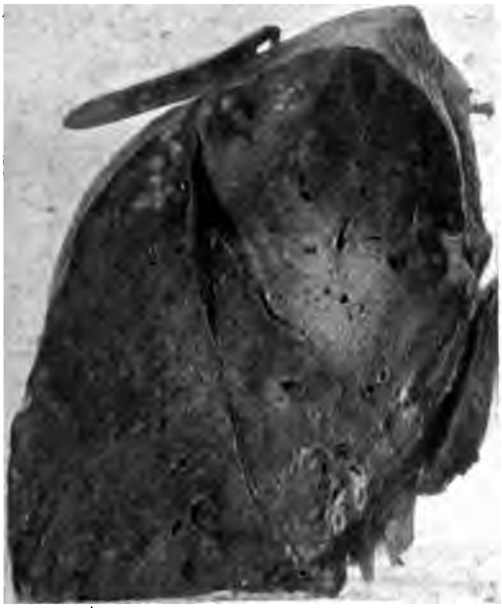


FIG. 235.—Specimen showing a small deposit of tubercles in posterior aspect of right per lobe and in the apex of the lower lobe. Corresponds to the right interscapular gion opposite fourth and fifth dorsal vertebra.

Following the involvement of the upper lobe the disease if progressing, anifests itself in the opposite apex; less commonly, according to Fowler, the upper lobe at a point corresponding with the apex of the axilla. isease of one apex with secondary manifestations in the opposite lower be occasionally occur.

One rarely finds during the terminal stage of the disease, that both ungs are equally diseased. The lung primarily affected is generally more extensively involved and the destruction of tissue, as manifested by cavity formation, greater. The lung, secondarily affected, may be markedly diseased in the upper lobe, but the lower will be free, or at most contain scattered tubercles. During the terminal months of the disease the lower part of one or the other lung, which undergoes a certain amount of compensatory emphysema, supplies the patient with most of his breathing space (Fig. 236; see also Fig. 101).

Another change, and one that probably follows the pulmonary infection very quickly, is a low-grade inflammation of the apical pleura which results in the obliteration of the pleural space at that point. While the clinical evidence of this is not great, it is an almost constant finding that in individuals who have died of chronic tuberculosis the apices of the lungs are firmly adherent to the chest wall and that this is most noticeable over the posterior surface.

As the pulmonary infection spreads the overlying pleural space becomes obliterated. Involvement of the pleura in tuberculosis is always greatest at the apex where the trouble originated (Fig. 237). At the apex the thickening is dense, gradually thinning out as the base of the lung is reached. While often dense throughout and binding the entire lung firmly to the chest wall, the adhesions over the lower part of the lung are more commonly lace-like and easily broken up. (See also section on Pleurisy, p. 572.)

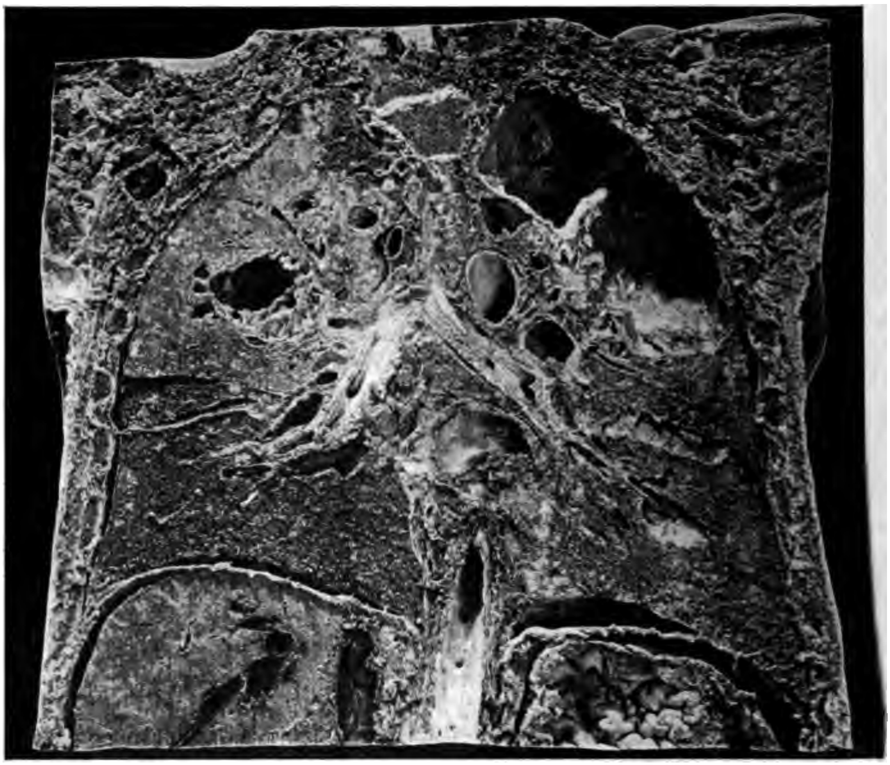


FIG. 236.—Large cavity in left upper lobe: dense infiltration and fibroid changes in lower lobe. Right upper lobe densely infiltrated with recently formed cavities. Right lower lobe practically free from disease.

Obliteration of the pleural space is, to a great extent, a protective measure. Were it not for this a pneumothorax would likely occur in the majority of cases of tuberculosis instead of being relatively uncommon. A more extended description of the changes which occur in tuberculous pleurisies and in pneumothorax will be found under the headings dealing with these conditions.

Dilatation of the bronchi is dependent on the amount of fibrous tissue which develops during the course of the disease. If the process is acute the bronchi present no change. On the other hand, in the chronic types of the disease with the formation of much fibrous tissue dilatation of the bronchi is not uncommon.

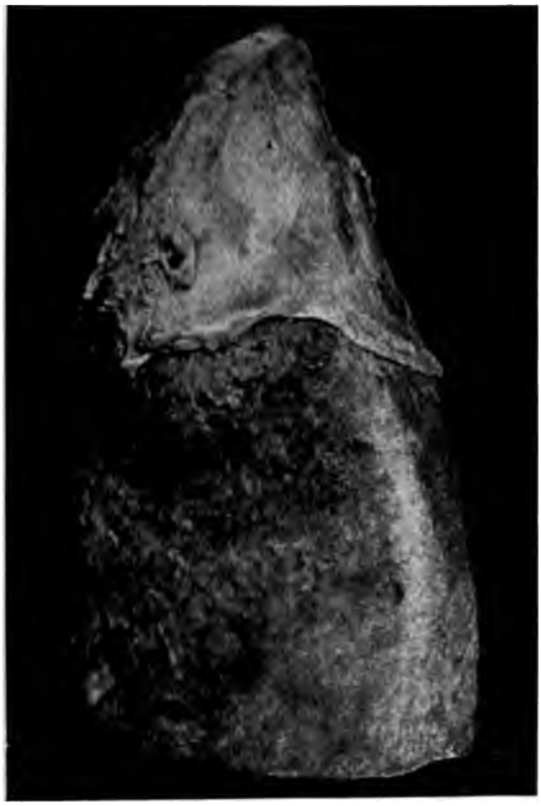


FIG. 237.—Thickened pleura at apex in tuberculosis.

Deformity of the chest and displacement of the heart depend largely on the chronicity of the disease. If chronic and there is much fibroid tissue present the affected lung shrinks and as a result the chest wall becomes retracted and the heart is drawn toward the diseased side.

Changes in Other Organs.—In addition to the characteristic changes which are seen in the respiratory organs, tuberculosis has a very serious damaging effect on other organs and tissues throughout the body. The brief account which follows is based on a study of 197 autopsied cases by C. Y. White.[1] The lesions most frequently noted are the presence of tubercles, usually the result of a terminal infection; congestion; catarrhal changes; fatty and fibroid degenerative changes and ulceration. In some situations all of these changes may be noted while in others one or more are apt to be present.

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[1] Fifth Report of Phipps Institute, 1909.

The *larynx* is frequently the seat of tuberculous changes in dying cases. Fetterolf[1] in a special study of 100 cases found the larynx involved in 83 and doubtfully so in 4. In the trachea, congestion is the most common change and in about 20 per cent. of cases, ulceration is present.

Many observers hold the view that the heart in tuberculosis is smaller than normal and deduce from this fact that the size of the heart bears some relation to susceptibility to the disease. Of 197 cases the weight of the heart, for both males and females, was practically that of the average normal heart. Cardiac changes noted at the autopsy table in tuberculosis are about the same as those encountered under other conditions. In about half the cases there are to be seen varying degrees of hypertrophy or dilatation, usually the latter, of one or more of the chambers. A small proportion show thickening of the valve leaflets and in a still smaller proportion, the valve orifices are incompetent or stenosed. Tuberculous infection of the heart muscle is rare. (See also section on Tuberculosis of the Pericardium, p. 656.)

Next to the lungs the *gastro-intestinal tract* and its associated organs are most seriously damaged. This leads to serious defects in metabolism, with consequent lowered nutrition. Some of the lesions are tuberculous, but most of them are of a secondary nature and because of the fact that they are so general and extensive, are far more serious to the functional activity of these organs than the lesions which are distinctly tuberculous in character. The intestinal tract is the site of ulcerative changes in from 80 to 90 per cent. of fatal cases. In some instances a large portion of the intestinal mucous membrane, especially the lower ileum, may be the site of ulcerative changes; in others there may be no more than three or four ulcers. The stomach is the site of an ulcer in about 2 per cent. of cases, which is about the usual incidence of gastric ulcer under all conditions. Rarely the ulcer is tuberculous in character. Pathological changes in the esophagus are unusual. Congestion of the mucous membrane is occasionally noted and in a few instances tubercle-like nodules are seen (3 out of 140 autopsies).

The secondary changes noted in the gastro-intestinal tract are subacute and chronic gastritis; gastro-enteritis, chronic catarrhal enteritis and colitis. Anatomical changes also occur. Gastroptosis was noted in 62 out of 197 cases; ptosis of the large gut was encountered in but two cases.

Tuberculous changes in the appendix were noted in 10 out of 197 cases. About 7 per cent. of the cases showed tuberculous fistulæ in the ischio-rectal region. Microscopical examination of the *ischio-rectal* region also showed the presence of a high percentage of tubercles in cases having no signs of fistula or abscess.

The parenchymatous tissue of the liver and pancreas usually shows widespread lesions varying from a slight cloudiness to complete necrosis or extensive fatty changes. Accompanying these lesions in the parenchymatous tissue there is generally some fibroid change. The same lesions are noted in the thyroid and suprarenal glands, both of which are lso concerned in metabolism. Amyloid changes in the liver are not ncommon.

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[1] Trans. Am. Laryngological Assoc., 1914.

Although the liver occasionally shows large caseous, tuberculous foci, his is rare. The presence of minute pinpoint and microscopic tubercles s very common and probably represents a terminal infection. The pancreas is quite resistant to the tubercle bacillus but even this organ occasionally shows microscopic tubercles. Tubercles are also noted in the thyroid gland and especially in the suprarenals. The latter may also be the seat of large caseous tubercles which may entirely destroy the organ.

The spleen is usually slightly enlarged and often congested. Miliary tubercles were noted in 60 and amyloid changes in 51 out of 197 cases.

Chronic inflammatory changes in *the kidneys* are extremely common but these changes do not differ essentially from those found in the kidneys of individuals dying of other chronic diseases. The changes are brought about by the extra work thrown upon the kidneys in the elimination of the toxic products generated by the tubercle bacilli or the organisms of mixed infections so frequently found. In addition to evidences of nephritis a very considerable proportion of the kidneys also show the presence of small miliary tubercles.

Other portions of the genito-urinary tract, in both the male and female, seem to be less liable to infection with the tubercle bacillus even in the terminal stages. Tuberculosis of the testicle rarely occurs in the course of the chronic ulcerative type of tuberculosis. When seen it usually dominates the clinical picture, the pulmonary disease being of secondary importance.

Involvement of *the meninges* is a not uncommon event in the chronic ulcerative type of tuberculosis. Meningeal tuberculosis is most often seen, however, in acute miliary tuberculosis. The condition is relatively common in children and it is very frequently the terminal event in cases of tuberculosis of the testicle.

Symptoms.—Pulmonary tuberculosis has no distinctive mode of onset nor is there any single symptom which is absolutely pathognomonic of the condition. In some instances there is a danger signal in the form of a single symptom; in the majority of cases, however, the onset is insidious and the disease may have caused considerable damage before its true nature is recognized.

A properly taken *history* should include information as to the occupation of the individual, the presence or absence of tuberculosis in the family, an account of past illnesses, and, lastly, but of the utmost importance, an account of the present illness. It is no exaggeration to state that from the history alone the vast majority of cases of early tuberculosis can be recognized definitely, or at least the presence of the disease can be strongly suspected. The mathematical chances of tuberculosis being present are overwhelming in the case of any individual who has an hemoptysis, an attack of pleurisy, a fistula in ano, or transient attacks of slight hoarseness, and this holds true in the face of previous good health and excellent physical condition. These single symptoms may be followed very shortly by other evidences of pulmonary disease. Very often, however, they are to be looked upon as prodromal symptoms, and months or years may elapse before the disease manifests itself in an active form. For one who is to become tuberculous the most fortunate thing is the occurrence of one of these single isolated symptoms, as they are apt to appear before there has been any deterioration in health and before the pulmonary lesion has become extensive. The significance of these manifestations is being constantly overlooked, partly because of the absence of other corroborative symptoms, but largely because of a paucity or even entire absence of physical signs.

The onset of pulmonary tuberculosis may be as follows:

1. *Hemoptysis and Blood-streaked Sputum.*—The one disease, above all others, in which hemoptysis most frequently occurs is pulmonary tuberculosis. This fact has been emphasized and reëmphasized in season and out of season and yet the true significance of this symptom is being overlooked constantly. Blood spitting is apt to occur in the great majority of cases of pulmonary tuberculosis at some time during the course of the disease. Among 5856 cases seen at the Phipps Institute the occurrence of an hemoptysis was acknowledged by 2790 (47.6 per cent.) at the time of their first visit. The difficulty, however, lies not with the self-evident case of phthisis but with those instances in which the blood spitting occurs as the initial symptom and before there has been any deterioration of health or the development of marked physical signs. It is extraordinary how frequently the source of the blood is ascribed to a bleeding point in the upper air passages in spite of the fact that there is ample proof that hemorrhage from this region is excessively rare. Although the apparent health of the patient and the absence or paucity of physical signs must be looked upon as strong factors in influencing physicians against a diagnosis of tuberculosis, the most important reason, in my opinion, is moral cowardice. Many will admit of a knowledge of the significance of a hemoptysis of obscure origin but in particular cases are unable to bring themselves to make a diagnosis without other corroborative symptoms and signs. In this connection Cabot's admonition is worth repeating: "I do not deny that the causes of hemoptysis are numerous, but I assert that the causes of *genuinely obscure hemoptysis* in temperate climates may be reduced to one—pulmonary tuberculosis." The objection may be raised that among the conditions which simulate tuberculosis most closely blood spitting is a very common symptom. So it is—and among such conditions may be mentioned the various mycotic infections, bronchiectasis, fibrosis of the lungs, malignant disease and distomatosis. The last-named has a fairly distinct geographical distribution and is to be encountered in this part of the world in imported cases only. Of the other conditions closer study will show in them certain differences, particularly in regard to the physical signs, which will often serve to distinguish them from tuberculosis. The real test, however, lies in the examination of the sputum. Cases presenting the symptoms and physical signs of tuberculosis without the presence of tubercle bacilli in the sputum should arouse at once the suspicion of some other origin for the hemorrhage. The main point is that a tentative or even a positive diagnosis does no harm in case the source of the hemorrhage is not a tuberculous infection. On the other hand, a negative diagnosis may lull the patient into a false security. There is little to be said in favor of the attitude of mind which favors withholding a diagnosis of tuberculosis in such cases because such an opinion, if wrong, places a stigma on the patient. Failure to give due heed to it more often ends in the disease progressing to hopeless incurability.

Figs. 238 and 239 clearly show the majority of the causes of hemopysis. That depicting Stricker's results deals with a body of men preumably healthy. It is apparent that the occurrence of blood spitting

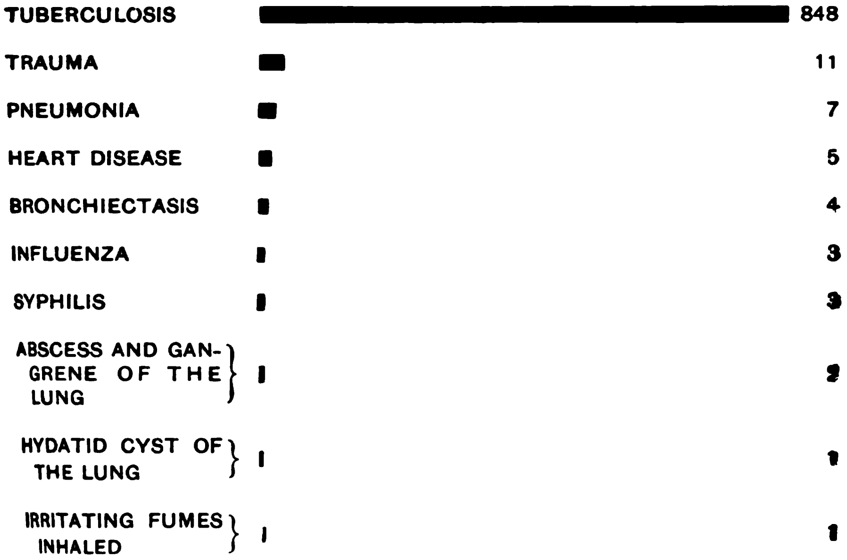


Fig. 238.—Causes of hemoptysis in Prussian soldiers. (*Cabot, "Differential Diagnosis," after F. Stricker.*)

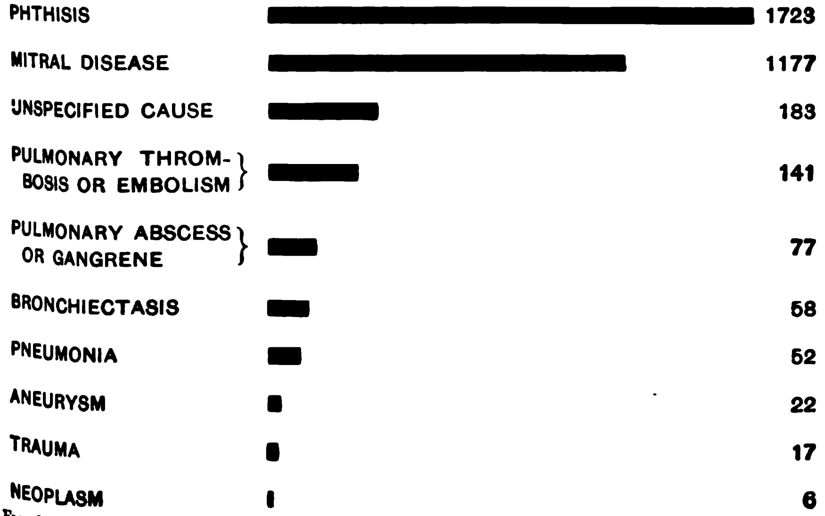


Fig. 239.—Causes of hemoptysis, Massachusetts General Hospital. (*Cabot, "Differential Diagnosis."*)

in such individuals points overwhelmingly to tuberculosis, the percentage in favor of this diagnosis being 95.8. The table compiled by Cabot from cases admitted to the Massachusetts General Hospital shows a wider distribution of the underlying causes.

It is to be borne in mind, however, that cases of obvious tuberculosis are not admitted to this hospital and that, as in the case with most hospitals, patients suffering from failing compensation form a very high percentage of the total admissions. Even so, tuberculosis occupies first place with a percentage of 50. Furthermore it is a fair inference that the majority of those classed as "undetermined" properly belong under the tuberculous group, which would raise the percentage considerably.

2. *Pleurisy.*—It is becoming more and more appreciated that an attack of pleurisy with or without effusion should arouse one's suspicion as to the existence of a pulmonary tuberculosis. At one time the occurrence of primary pleurisy was looked upon as relatively common. The attack may be without an apparent exciting cause or it may follow exposure to cold. In any event it is now recognized that at least 80 per cent. of such pleurisies are tuberculous in origin. A pleural effusion which develops insidiously and attains a large size before recognition is almost certainly tuberculous.

The records of the large insurance companies show that the death rate from tuberculosis of the lungs among persons who have had pleurisy within five years prior to insurance is three times the average rate for individuals without such a history.

3. *Ischio-rectal Abscess and Fistula in Ano.*—Proctologists have performed a valuable service in emphasizing the tuberculous nature of the great majority of abscesses in and about the ischio-rectal fossa. The occurrence of a pyogenic infection in this region should always call for a careful physical examination of the chest. In the event of a negative examination the patient should be warned as to the possible danger and cautioned as to his mode of life. Very often the ischio-rectal abscess will precede definite pulmonary symptoms by several years.

4. ~~*Hoarseness.*~~—This may be the very first intimation of pulmonary tuberculosis. The hoarseness may develop without definite exposure to cold or any other known cause. It may be persistent from the onset but more often is transient in character, being present for a few days and then disappearing and reappearing again. Very often the hoarseness is noticeable for a part of the day only; either on getting up in the morning or late in the day or after much talking. A laryngological examination is imperative in these cases. Nothing is more fatal than to assume that the laryngitis is due to a "cold."

The occurrence of any one of the above-mentioned symptoms furnishes the most valuable information possible as to the presence of a pulmonary tuberculosis. Unless it can be shown conclusively that they are produced by something else there should be no hesitation in making a diagnosis of tuberculosis. Furthermore, it cannot be too strongly emphasized that the physical appearance of the patient should have no influence in arriving at a conclusion. Only too frequently physicians hesitate to commit themselves because the patient presents every appearance of health and the pulmonary signs are indefinite.

Unfortunately tuberculosis is far more often characterized by an insidious rather than an abrupt onset, and there is no single, striking symptom upon which we can place much reliance. Cases with an insidious onset may manifest themselves in a variety of ways.

1. The individual catches "cold" which instead of clearing up persists, or just as it seems to be getting better a fresh "cold" is acquired.

Associated with the cough, which may be dry and hacking in character, there are loss of weight, anemia, malaise and at times the sputum may be blood-tinged. In some instances patients themselves neglect these "colds," in other instances although they seek advice, they are told they are suffering from bronchitis.

It cannot be sufficiently emphasized that a cough which, without complications, persists over six weeks or two months is suspicious at least; if it is associated with loss of weight, slight fever, and malaise, it is almost certainly tuberculous in origin.

2. In another group the dominant symptoms are referred to the *gastro-intestinal tract*. Anorexia is common, or the appetite is variable, being sometimes very good and at others, poor. Symptoms of indigestion are annoying and usually take the form of distention after eating, with a sense of discomfort or even well-marked pain in the epigastric region. Constipation is usually present. In this group malaise, pallor and loss of weight also occur. Cough may be present or absent. If present it is usually not marked and its significance is lost sight of, because of the marked gastric symptoms. Stomach coughs were once common, but fortunately little is heard of them nowadays. Indigestion occurring in an individual previously free from any such disturbance and associated with malaise, loss of weight and cough, should lead one to take a careful history of the case and above all make a thorough examination of the lungs.

3. A third group is comprised of those cases in which the onset is so insidious that there is nothing which definitely attracts attention. The condition is often described by the expression "generally run down." A careful analysis of the symptoms in this group usually reveals the following facts: Malaise is marked and the individual has a feeling of being constantly tired, even when awakening after a good night's sleep; there is loss of weight which may amount to 10 or 15 pounds, so gradual, however, that the patient has not been conscious of it; there is usually some pallor, which is more marked in the morning; cough may be absent, or if present, is confined to the morning on awakening; and lastly, there may be occasional attacks of indigestion. The most significant features are the malaise and gradual loss of weight. Patients in this group frequently escape detection in the incipient stage of the disease.

In women disturbances of the *menstrual function* are among the early symptoms. Either the flow becomes scanty and irregular in its appearance or it ceases entirely. Under the same circumstances leukorrhea may appear or if already present become greatly aggravated.

4. The attack may begin more abruptly and resemble in every respect *typhoid fever*. The patient feels tired and dragged out and there is slight fever which finally assumes the continuous type seen in typhoid patients. Inasmuch as bronchitis and some cough are commonly present in typhoid fever patients the true nature of these symptoms are overlooked. I recall one patient who was treated for nearly three weeks for typhoid fever, without a suspicion as to the true nature of his trouble, until he had a fair-sized pulmonary hemorrhage following a cold bath. This patient had a moderately advanced lesion in the right upper lobe and tubercle bacilli in the sputum.

I think there can be but little doubt that tuberculosis not infrequently undergoes an acute exacerbation which closely resembles an attack of typhoid fever. Very often the acute process spontaneously subsides and the patient apparently makes a perfect recovery. Out of 5895 cases of tuberculosis seen at the Phipps Institute 1083 or 18.3 per cent. gave a history of having had typhoid fever. It is quite likely that not a few of these were instances of an acute exacerbation of tuberculosis.

5. Occasionally tuberculosis first manifests itself with chills, fever and sweats which may assume a periodicity similar to that seen in *malaria*. In regions where both diseases coexist mistakes are frequent.

6. What is known as *Louis' law* should be borne in mind, namely, that after the age of puberty a tuberculous lesion in any part of the body is almost invariably accompanied by pulmonary tuberculosis. Therefore, the presence of a tuberculous testicle or of tuberculous lymph nodes in an adult should suggest a careful examination of the lungs.

7. Finally, the following precepts of Dieulafoy should be kept in mind: Every youth or adult, who wastes much or rapidly, with or without fever, must be suspected of having tuberculosis, in the absence of diabetes or Basedow's disease.

Every girl or young woman who has neither genuine chlorosis, Bright's disease nor syphilitic anemia, but yet has the appearance of chloro-anemia, must be suspected of having tuberculosis.

The symptoms detailed above in the various modes of onset also form the symptomatology of the incipient stage. If the disease is arrested at this stage the symptoms disappear. If, however, the disease progresses the symptoms already present are apt to become more marked; the emaciation becomes more pronounced, the cough and expectoration more troublesome, and in addition new symptoms are constantly developing. By the time the terminal stage is reached the patient will suffer at one time or another from all of the symptoms peculiar to the disease.

The symptoms of tuberculosis are of two kinds: (1) the general or constitutional symptoms due to the toxic effects of the infection; and (2) the local subjective symptoms due to pathological changes in the various organs.

Constitutional Symptoms.—*Fever.*—This is the most important symptom in tuberculosis and a study of its manifestations yields much information, as from this one symptom it is possible to trace with reasonable certainty, the destructive process of the disease as it progresses from its incipiency to the terminal stages. On the other hand, the subsidence of the fever is the most reliable evidence we have that the disease is tending toward arrest or quiescence.

The early febrile manifestations of tuberculosis usually consist of slight elevations of temperature amounting to from 1° to 2.6°F. The fever may be present every day or only every second or third day (see Fig. 240). It is increased by exercise and as a rule quickly subsides after several weeks' rest in bed. At times slight fever persists in an early case even after prolonged rest (see Fig. 241). This is usually an indication of poor resistance or the presence of more extensive disease than the physical signs indicate.

Very often the febrile stage is succeeded by a period of subnormal temperature during which time the patient continues to improve (see Fig. 242). Gradually the temperature curve shows less marked remissions and becomes normal. Unless a relapse occurs or there is some intercurrent disturbance the temperature then remains normal. Sub-rmal temperatures are also present in cases with advanced disease ich is temporarily quiescent. In such instances the vitality is usually or.

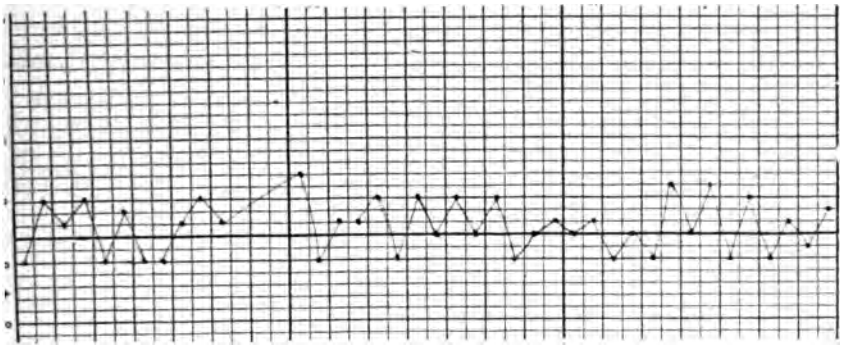


FIG. 240.—Temperature in an early case with lesion at right apex.

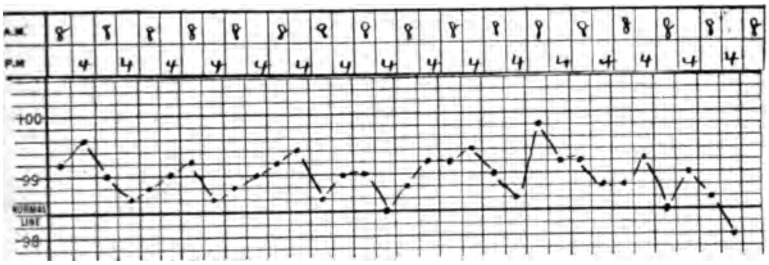
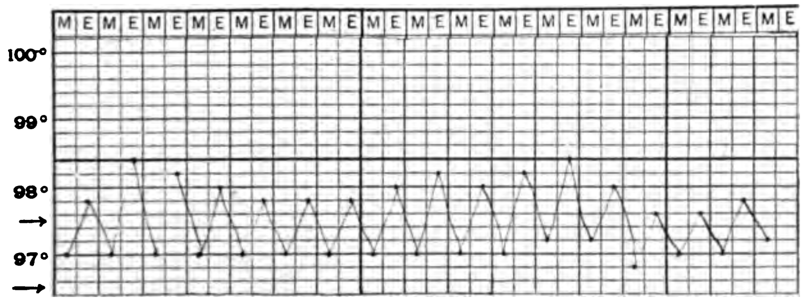


FIG. 241.—Persistent slight fever in an early case after prolonged rest in bed.



242.—Subnormal temperature often noted after subsidence of fever in early case.

A pulmonary hemorrhage may be followed by fever. This may be e to an acute respiratory infection which has been the exciting cause of hemorrhage or it may result from small areas of broncho-pneumonia e to insufflation of blood.

In persons who have become afebrile, an abrupt rise in the temperature may occur and last for a day or so. This may be due to an acute cold (see Fig. 243), some gastric disturbance, constipation, slight pleurisy, overexertion, etc. In women during the active stage of the disease the temperature may abruptly rise some days prior to, or with the onset of, the *menstrual period* (see Fig. 244) and may remain elevated until the flow ceases. In patients who have been afebrile, and in whom these temporary manifestations of fever occur, it is usually an indication that the disease has not become arrested but is simply quiescent. When they can pass through such episodes without a febrile reaction, it may be taken as one of the pieces of evidence that the disease is apparently arrested.

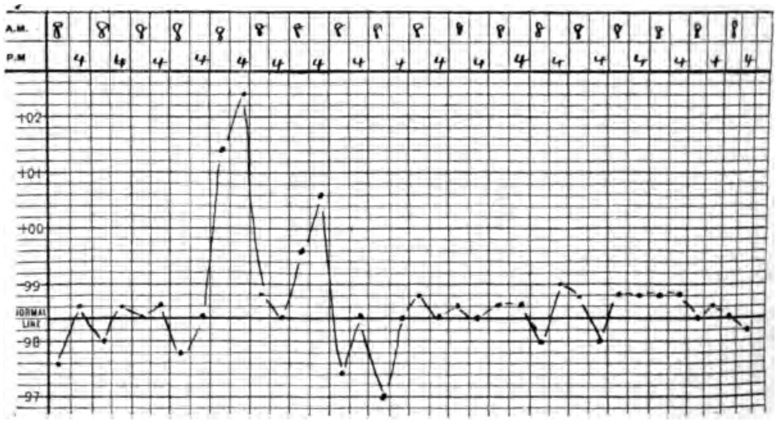


FIG. 243.—Afebrile case. Rise in temperature due to an acute cold.

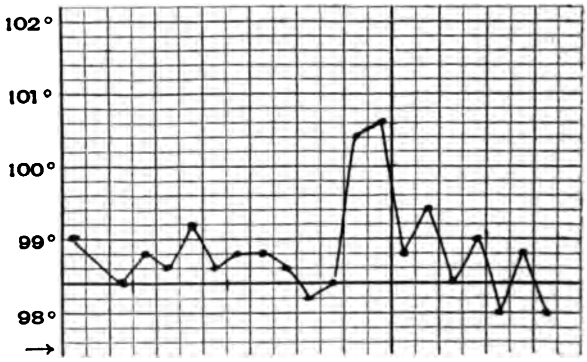


FIG. 244.—Rise in temperature due to menstruation.

When the disease is not arrested but continues to spread the temperature is moderately high and may be intermittent, remittent or continuous (see Fig. 245). The temperature may then subside and become normal or show but slight elevations or it may pass into the hectic type which is characterized by marked remissions and intermissions

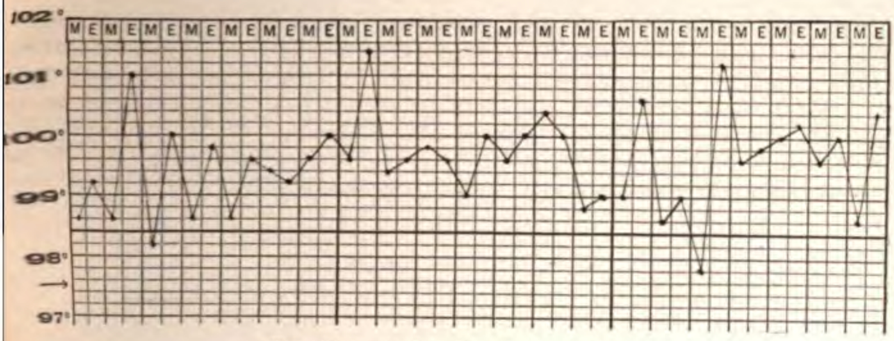


FIG. 245.—Active disease progressing.

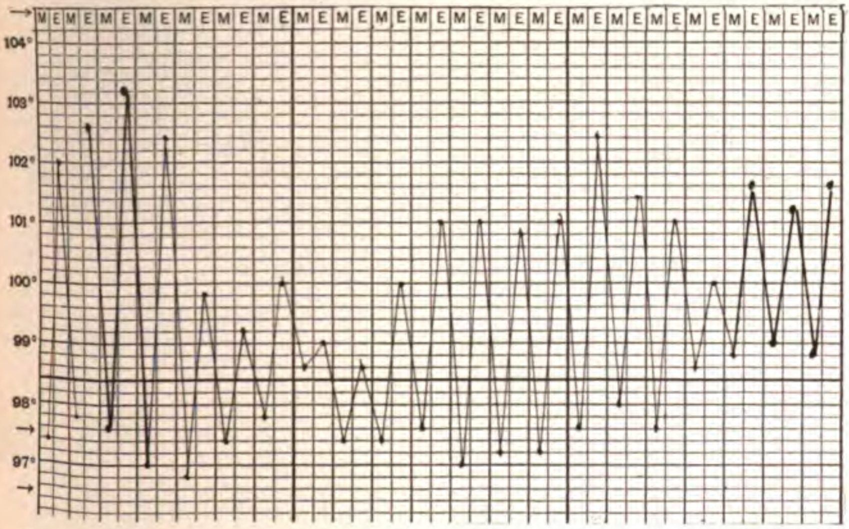


FIG. 246.—Intermittent and remittent type of temperature seen in advanced cases.

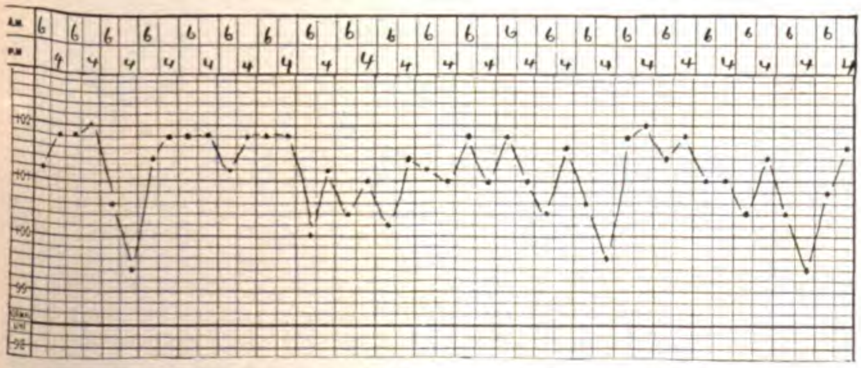


FIG. 247.—Rapidly advancing process of bronchopneumonic type. Also shows inverted temperature.

(Fig. 246). In this type of temperature, which is always present in advanced cases which are progressing toward a fatal termination, the difference between the morning and evening temperature may amount to from 5° to 8°F.

When new areas of lung tissue are being invaded the temperature is continuous in type, sometimes with slight remissions and in other instances there are marked intermissions (Figs. 247 and 248). Such temperature curves always indicate marked activity of the disease. Temperatures of this type may be present when the case is first seen or develop after a period of slight fever; as a rule they change into the hectic type but the fever may gradually subside and the temperature become normal. Another interesting feature to be looked for in charts of this type is inversion of the temperature, that is the morning temperature is higher than that in the evening. An inverted temperature is com-

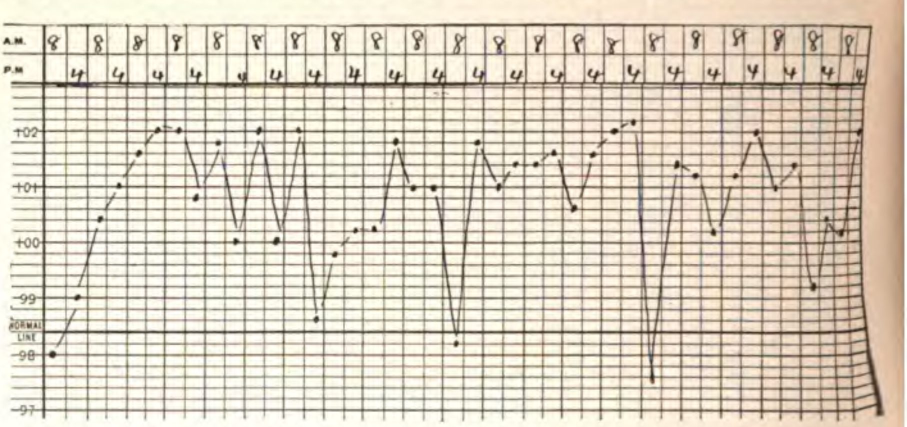


FIG. 248.—Rapidly advancing type of disease. Also shows inverted temperature.

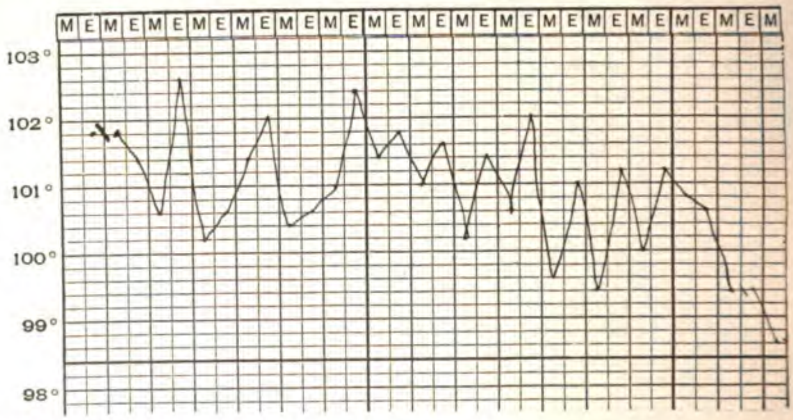


FIG. 249.—Temperature curve resembling that of lobar pneumonia. In this case the temperature fell to normal in three weeks.

monly associated with miliary tuberculosis but it often may be noted in pulmonary cases in which the disease is spreading rapidly. It is not constant and is usually observed but a few days as shown in both Figs. 247 and 248.

Another type of temperature is that seen in cases with an acute onset and high fever. The fever may subside by lysis in two or three weeks (see Fig. 249) or it may become hectic in type. Such cases are not infrequently mistaken for *lobar pneumonia* because of the sudden onset, high fever, blood in the sputum and signs of consolidation over an upper lobe.

A continuous type of temperature which is broken at intervals by marked intermissions (Fig. 248) sometimes leads to a diagnosis of *malaria*, especially when the sudden rises in the temperature are accompanied by chilly sensations.

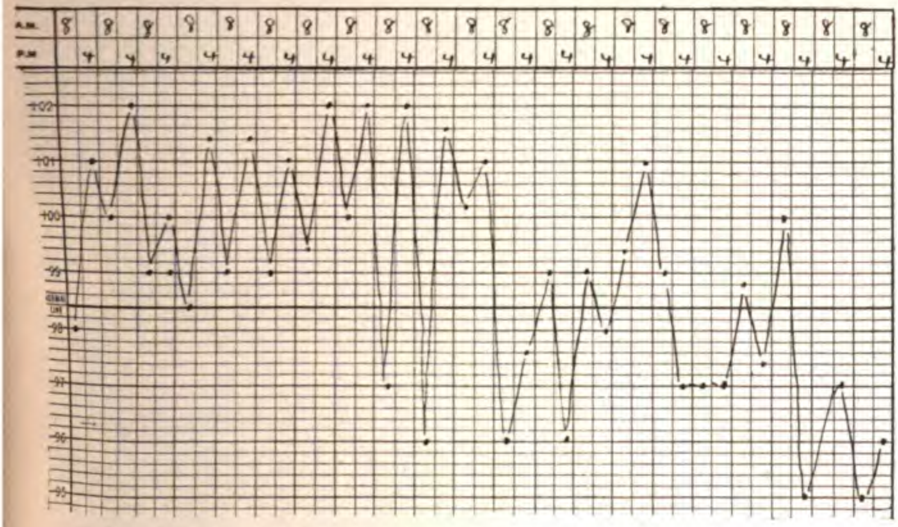


FIG. 250.—Type of temperature seen in terminal stage of advanced cases.

In the terminal stage of the disease the temperature is characterized by marked irregularities, such as shown in Fig. 250.

The *diagnostic and prognostic value of temperature* observations cannot be overestimated. The clinical thermometer should always be used. In the very early cases when the symptoms and physical signs are inconclusive, the use of the thermometer three or four times a day for a week, will often determine the diagnosis. Furthermore, the absence cases with other unequivocal evidences of the disease, or the dence of fever are the best evidences we possess of an inactive use. The temperature should always be taken in the morning the afternoon, preferably at 4 P.M. Additional observations le at noon and 8 P.M. if thought necessary.

n the use of the clinical thermometer is a simple procedure utions should be observed. Not infrequently I have been told that a patient had no fever when subsequent observations in a sanatorium showed that there was a slight rise in the temperature every afternoon. In order to detect slight rises in the temperature (99°, to 99.3°F.) the thermometer should be left in the mouth not less than 10 minutes. For temperatures over 100°F. any thermometer will register the amount of fever within a minute, but for slight elevations even the half minute and minute thermometers will often fail. Several years ago I carried out a series of observations which showed that a 3-minute thermometer, if kept in the mouth for at least 10 minutes, was for practical purposes, as accurate as the quickly registering thermometers placed in the rectum. Mouth temperatures should not be taken within half an hour if the patient has taken anything very hot or very cold to eat or drink.

Many observers recommend the rectal use of the thermometer as the only reliable method. If the precautions I have given are observed this will not be necessary. Besides it is less convenient and often patients object to it.

Loss of Weight.—A gradual and progressive loss of weight is characteristic of the disease. The amount lost varies greatly in different individuals and is governed to a large extent by the severity of the infection. In the acute types of the disease the loss is rapid and may eventually amount to a third or more of the normal body weight. In the chronic forms of the disease the loss of weight is rarely extreme and in addition it is apt to be quickly regained if the patient is put under the proper treatment. A progressive loss of weight which continues in spite of proper treatment is always of serious import. On the other hand, a steady gain in weight is one of the most favorable prognostic signs.

Not only does the patient show emaciation but the muscular system becomes more and more atrophied as the disease progresses. In addition the muscles become unduly irritable.

Malaise.—In many cases with an insidious onset a sense of weariness with or without exertion of any kind may be the first manifestation of the disease. Very often it is the only symptom the patient complains of for months. It is the one symptom that has walked more patients into their graves than any other because of the fact that exercise is almost invariably prescribed. This is commonly done without attempting to find out whether there are any associated symptoms. The exercise is prescribed on the ground that inasmuch as it makes a healthy man feel better it ought to make the individual who is suffering from languor feel twice as well. Unfortunately this procedure aggravates the difficulty instead of relieving it. As the disease progresses the sense of languor and tiredness increases. In the terminal stages of the disease it is due to the extreme emaciation and inability to absorb the proper amount of nourishment.

Closely allied to this condition of general malaise is *neurasthenia.* It is becoming more and more recognized that neurasthenia is rarely a pure functional disturbance. In some cases of tuberculosis, neurasthenia is a very marked feature of the disease and the nervous phenomena may dominate the picture. On the other hand, there are many cases of neurasthenia which are considered as instances of a functional disturbance of the nervous system but which in reality are due to a latent tuberculous infection which escapes notice. I recall one case in which neurasthenia, associated with profound asthenia, had existed for four years. Although the neurologist in charge suspected a tuberculous infection it could never be located. Finally the occurrence of an hemoptysis pointed to pulmonary trouble which a careful physical examination showed to be present in the apex of the right lung. The patient was then treated as a case of tuberculosis and made a complete recovery.

Night sweats may occur at any time during the course of the disease but are not usually encountered in the incipient stage. They occur very commonly in the second stage but as a rule are not severe. Sweating varies greatly in different individuals. In some cases it is the most distressing and harassing manifestation of the disease, as it is not only exhausting but in addition causes great discomfort. As a rule night sweats, which do not disappear after the patient has been put under treatment, occur in the acute types of the disease and in the terminal stages of the chronic form when fever is present. Sweating rarely occurs in an afebrile patient. Night sweats, as the name implies, occur at night, usually in the early morning hours.

Axillary sweating is very common in tuberculous subjects even in the incipient stage of the disease. A tendency to sweat after the least exertion is also of frequent occurrence during the early stages of the disease.

Chills.—Sometimes chills occur as an early manifestation of the disease. As a rule chills associated with fever and sweats, occur in very acute types of the disease, such as pneumonic phthisis and acute miliary tuberculosis. In the terminal stage of chronic ulcerative tuberculosis, while chilly sensations are not infrequent, distinct chills are unusual.

Anemia.—A slight degree of anemia in the incipient stage of tuberculosis is not uncommon, although, as a rule, it is more apparent than real. Morning pallor is often very noticeable; this is apt to be masked in the afternoon by flushing of the cheeks as the result of slight fever.

In the second and third stages of the disease anemia may or may not be present. Sometimes it is quite marked. In other instances and especially in individuals who have been under appropriate treatment, there is no evidence of anemia; on the contrary, the patients often present a healthy, ruddy color.

The blood picture in tuberculosis has a slight resemblance to that seen in chlorosis in that the hemoglobin is most affected, although the reduction of the hemoglobin content in tuberculosis is never as marked as in chlorosis. The leukocytes in tuberculosis are not increased in the first and second stages; in the third stage a moderate leukocytosis is common, probably as the result of mixed infection.

Gastro-intestinal Symptoms.—As already mentioned these symptoms may overshadow the pulmonary symptoms at the onset of the disease. Anorexia is not uncommon in the beginning and may persist until the end. In some patients gastric disturbances persist or recur very frequently throughout the course of the disease and seriously interfere with recovery. The majority of people suffering from tuberculosis give both subjective and objective evidence of the injury which befalls the alimentary canal and the organs which have to do with nutrition. A coated or dry tongue, loss of appetite, nausea and vomiting, constipation, and diarrhea are the common symptoms which are encountered. One or the other of them exists in almost every case. As a general rule it can be said that the chances of recovery are very poor if the patient either cannot eat because of marked anorexia or because the least thing upsets the stomach. In spite of extensive disease of the lungs and marked symptoms some patients continue to have a good appetite and digest their food without discomfort. In those who suffer from a sense of fulness, often with considerable epigastric pain after eating, the trouble is rarely due to pathological changes in the stomach but to alterations in the character of the gastric juice. The gastric juice may be inefficient because it is too small in amount or too poor in quality. At times nausea and vomiting may occur but more often the patient simply feels nauseated either at the sight of food or after eating. Vomiting more often results from a reflex action as the result of a paroxysm of coughing. In the effort to build the patient up forced feeding is sometimes injudiciously employed with the result that the overfeeding produces a typical bilious attack.

Diarrhea is sometimes a very troublesome symptom. It may be due simply to a catarrhal inflammation of the intestinal mucous membrane or as the result of intestinal ulceration. It may be stated, however, that diarrhea even when most severe, cannot be taken as an indication of tuberculous ulceration of the intestines. Recently Brown and Heise[1] have shown by means of a bismuth meal that there is marked hypermotility of the intestines when ulceration is present, the gastro-intestinal tract becoming entirely emptied within 24 hours.

Cardio-vascular Symptoms.—Acceleration of the pulse is a very frequent manifestation in tuberculosis. It may occur as an early symptom and without fever. Under these circumstances, it is of value as an early diagnostic sign. As the disease progresses the tendency of the pulse rate to increase becomes more marked. In some instances the patient is not conscious of the rapidly beating heart but at times an annoying palpitation occurs with the tachycardia. During the last stage of the disease the pulse rate is usually very high rising to 120 or more.

The *blood-pressure* in tuberculous subjects is usually low. If the disease undergoes arrest and health is restored, the blood-pressure returns to normal. Cardiac weakness is not uncommonly present and occasionally is the cause of death.

Edema of the extremities, especially the legs, is not infrequently seen in the terminal stage of tuberculosis. It is always an indication that the end is near at hand. Edema may be due to cardiac weakness, a complicating nephritis or both.

A violet or bluish discoloration of the nails is frequently noted. When it occurs in the early stages of the disease it is usually an indication that the infection is a serious one. It is almost invariably present in the terminal stage. While the condition resembles cyanosis due to cardiac weakness it occurs independently of the heart and is apparently due to toxic absorption. True cyanosis of the fingers and lips is occasionally noted.

Skin.—In the early stages of the disease the skin rarely shows any abnormality. In the advanced stages, however, it is nearly always unduly dry or moist and clammy. Often the skin has a scaly appearance and fine bran-like flakes can be scraped off. In those who are uncleanly in their habits pityriasis versicolor, an eruption which consists of yellowish or orange-colored patches, is commonly seen on the thorax and upper abdomen.

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[1] *Jour. Amer. Med. Assoc.*, July 12, 1919.

Jaundice and purpura are occasionally noted in tuberculosis; usually in the terminal stage. In an analysis of the ward patients in the Phipps Institute, Cruice found 7 instances of jaundice among 1748 cases and 8 of purpura among 1626 cases.

A hectic flush is common in the afternoon even in patients who have a very slight rise in the temperature. The flush is usually confined to the side affected or if both lungs are involved will be more marked on the side having the most marked disease.

In advanced cases the hair becomes thin and presents a dry, lusterless appearance.

Urine.—In the early cases evidences of kidney irritation occur with no greater frequency than is encountered in the general run of individuals. As the tuberculous process advances, however, the urinary findings show an increasing number of abnormalities. Walsh found albumen present in 47 per cent. of advanced cases and casts are nearly as frequently present. In a small percentage of cases sugar is present. It was noted in 2 per cent. of 656 cases studied at the Phipps Institute. In some instances it is due to a true diabetes; in others it is to be looked upon as a mild glycosuria.

Ehrlich's *diazo reaction* was considered at one time to be of both diagnostic and prognostic value. As it is often found in conditions other than tuberculosis, it has no significance as a diagnostic sign; and the same may be said of its prognostic value.

Phosphaturia is often present when the patient is steadily losing weight. It is looked upon as an evidence of cell destruction.

Nervous System.—The *mental attitude* varies greatly. This, however, has little to do with the disease itself; for while it is true that most people are depressed at the onset, they rapidly revert to their normal state of mind. This may be optimistic, pessimistic or indifferent as the case may be. It is a common belief that the *spes phthisica* is of frequent occurrence in tuberculous subjects. It is extraordinary how some patients will cheerfully make plans for the future even when they are a few days from death. Although the frequency of this exaggerated form of hopefulness has been greatly overestimated, it is undeniably true that cheerfulness and optimism as to the outcome, is more often present than depression and pessimism.

Tuberculosis among the insane is very common but no more so than a similar group of individuals living under like circumstances. On the other hand the development of a psychosis in a tuberculous subject is relatively infrequent. McCarthy recognizes a small group of psychoses due to tuberculosis and a second group which includes the usual type of insanity determined by a lowering of nutrition. The first group presents a symptom complex closely resembling paresis. In the second group the psychosis may take the form of melancholia, mania, dementia præcox, hysterical insanity and delusional insanity. Suicidal tendencies are not uncommon especially in those who become depressed and melancholy.

Hyperæsthesia.—Points of tenderness may occur in various parts of the body and in some instances there is a definite neuritis.

Herpes zoster is occasionally noted in tuberculosis but it is doubtful whether the condition can be ascribed to the tuberculous infection. Headache is not infrequent. It is commonly due to eye strain and is usually encountered in those confined to bed and who read a great deal. Persistent headache should always arouse suspicion as to the presence of a meningitis.

Insomnia of a mild grade is not uncommon; it is rarely severe enough to demand treatment. The sleep is often broken, however, by night sweats or attacks of coughing.

Arthritis, generally affecting the ankles, is in my experience relatively common in advanced cases. The ankles become slightly swollen, reddened and very painful. In some instances the pain is intense and even the weight of a sheet is unbearable. A number of French observers look upon this type of arthritis as being tuberculous in origin.

Menstrual Function.—In women the menstrual function is affected sooner or later, in the majority of cases. Sometimes suppression of the menses is one of the earliest manifestations of the disease. As a rule, the menstrual flow first becomes irregular and scanty and finally ceases. If the patient is restored to health the menstrual function gradually becomes normal. Leukorrhea is also apt to develop or if present it becomes much worse.

Local Subjective Symptoms.—*Cough.*—In the majority of cases cough is among the very earliest manifestations of the disease and is usually the first symptom that the patient notices. At first it may be confined to the morning on awakening or it may be absent at that time and occur intermittently during the day. In still other instances it is most marked on going to bed. Not infrequently what is at first believed to be an acute cold continues indefinitely. It is a safe rule to view any cough which persists for six weeks or two months as being tuberculous in origin unless the contrary can be proved definitely. As the disease progresses the cough becomes more and more severe and tends to become paroxysmal in character.

Occasionally the patient has a number of symptoms indicating the existence of an incipient tuberculosis and no cough whatever. The cough is due to the irritation of the bronchial mucous membranes by the pulmonary secretions and is almost invariably relieved by the expulsion of the sputum. Any cough which is unproductive is largely unnecessary and can be controlled voluntarily to a great extent by the patient.

Expectoration.—As a rule the cough and expectoration are closely associated and the one is almost invariably accompanied by the other. There may be considerable expectoration, however, with little or no cough, the sputum being brought up by what is known as hawking or clearing the throat. In the early stages of the affection the sputum is usually slight in amount but as the disease progresses it becomes more and more profuse, especially after the formation of a cavity.

As to the sputum in tuberculosis it can be said that it has no distinctive characteristic, except possibly in the stage of excavation, when the so-called nummular sputum occurs. The latter is brought up in the form of irregularly shaped grayish or greenish-gray balls which sink in water. Sputum with similar characteristics may occur also in bronchiectasis.

Aside from this form of sputum the secretion may consist of glairy transparent material with black specks scattered through it; it may be slightly or markedly yellowish in color and later it may assume a greenish tinge. From time to time blood may be noted in the sputum. The blood may occur in the form of a few minute specks; it may be in the form of streaks or the blood may be intimately mixed with the sputum giving it a pinkish or reddish color, depending on the amount of blood present.

Hemoptysis.—The significance of hemoptysis has been alluded to in considering the modes of onset. Hemoptysis occurs in about one-half of all cases of pulmonary tuberculosis, although some observers have placed the incidence as low as 30 per cent. and others as high as 80 per cent. Of 5856 cases observed at the Phipps Institute 47.6 per cent. had had an hemoptysis at some time during the course of the disease.

Males are somewhat more liable to hemoptysis than females, probably because of their greater physical activity. The accident occurs most frequently in adults who are suffering from the chronic ulcerative type of disease. Hemoptysis is an unusual occurrence in children nor is it often noted in the acute types of the disease in adults.

The amount of the hemorrhage varies greatly. It may consist of a few flecks or streaks of blood in the sputum or it may be so large as to cause death within a few minutes. The sputum may be constantly streaked with blood or pinkish or salmon-colored for weeks or even months. When the hemorrhage consists of an ounce or so of pure blood the patient may expectorate dark clots or blood-streaked sputum for a few days. A single hemoptysis of this kind may be the only manifestation of blood the patient ever has. Very often, however, there are recurrences at more or less frequent intervals. Very large hemorrhages almost invariably occur in the advanced stages of the disease when cavity formation has taken place. As a cause of death hemorrhage is not as frequent as ordinarily believed. In a study of the cause of death in 136 cases I found that a hemorrhage was the immediate or exciting cause in 11. The hemorrhage may be so large as to immediately cause death or it may give rise to a widespread broncho-pneumonia due to insufflation of blood, which proves fatal in a few days.

In the early stages of the disease the source of the bleeding is usually from the pulmonary veins and the blood is bright red in color due to the fact that the pulmonary veins carry arterial blood. In the moderately advanced and advanced cases the blood comes from the pulmonary artery and is dark or venous in color.

In many instances the exciting cause of the bleeding can be determined but in not a few cases it suddenly appears without apparent cause. Recently I saw a man who had been discharged from the White Haven Sanatorium ten years ago with his disease arrested. During all this time he had been in perfect health and had worked at his trade every day. While walking along the street he suddenly was seized with an attack of coughing and spat up a few ounces of blood. Aside from this he had no other symptoms and examination of his chest shows no change from that noted ten years ago.

In many instances the exciting cause of the bleeding is an acute respiratory infection commonly manifesting itself as an ordinary "cold." In institutions it is not at all unusual for a number of patients to have attacks of hemoptysis within a few days. This epidemic-like occurrence is undoubtedly closely related to some infection. Some years ago Flick, Ravenel and Irwin in a study of such cases were able to show the almost constant presence of pneumococci in the expectorated blood.

As an exciting cause of pulmonary hemorrhage exertion of some description is usually considered as a common factor. It is interesting to note that Bang[1] in a study of hemoptysis found that the hemorrhage came on while the patients were lying in bed or reclining in a chair in 69 per cent. of 354 cases. He considers congestion or stasis to be the most important factor. Thus 50 of the patients had been constantly febrile; fever had developed just prior to the hemorrhage in 45 and immediately afterwards in 31; 11 had been severely chilled and 10 had received an injection of tuberculin.

Among women blood spitting may be present just before or during the time of the menstrual period. As the disease tends towards arrest, the bleeding gradually ceases.

In cavity cases large hemorrhages are usually the result of rupture of a pulmonary blood-vessel. In a large percentage of cavities there are present in the walls or in the trabeculæ traversing the cavity, blood-vessels. Small aneurismal dilatations are very commonly present in the vessels. They may rupture through a sudden elevation of the blood-pressure or the disease may gradually erode through the arterial wall. In addition to hemorrhages of considerable size the sputum from cavity cases may be pinkish or salmon-colored from the admixture of blood coming from ulcerations in the wall of the cavity and such sputum may be the precursor of a large hemorrhage.

Pain.—Chest pain is one of the common manifestations of tuberculosis. The character of the pain varies. It is usually subjective but may be objective only. When objective it is usually associated with cavity formation in the lungs and is noted in percussing over the site of the cavity. Often there is no subjective evidence of pain in spite of an extensive pleural friction rub. In such cases pain can usually be elicited by pressure with the finger tips over the site of the friction.

The character of the subjective pain varies greatly. It may be sharp and knife-like as the result of a severe pleurisy or in other instances it may consist of a feeling of soreness. Not uncommonly the pain or soreness is referred to the region of the shoulder on the affected side. This fact should be borne in mind as it is commonly mistaken for a rheumatic manifestation.

One of the severest forms of pain occurring during the course of tuberculosis is that sometimes caused by pneumothorax. The pain may be the only indication that such an accident has occurred. The great majority of tuberculous patients have some manifestation of chest pain at one time or another during the course of the disease. Among 3007 cases at the Phipps Institute chest pain was noted in 2280 or 75.82 per cent.

Vague and indefinite pain which has no fixed location and no apparent anatomical basis to explain its cause is frequent in neurasthenic patients. Among dispensary patients of Jewish birth, it is to be met with constantly.

Hoarseness.—Laryngeal involvement is very common in pulmonary tuberculosis. At one time it was looked upon as an absolutely hopeless complication. This conception, however, took into account only advanced laryngeal lesions. We now know that the laryngeal process develops gradually and has an incipient, a moderately advanced and an advanced stage. Furthermore, just as in the lungs, the type of the lesion as considerable bearing on the outcome. From the clinical standpoint he essential thing to bear in mind is the significance of hoarseness.

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[1] *Ugeskrift for Laeger*, March 23, 1916.

Hoarseness as we have already pointed out may be temporary or ersistent. When temporary it may be constant for a few weeks and radually disappear, especially if use of the voice is prohibited. In ther instances it occurs only in the morning on awakening or late in the ay if the voice has been used. Persistent hoarseness is usually indicaive of serious damage to the larynx; it may be associated with advanced ulmonary lesions or may itself constitute the major lesion.

The great majority of fatal pulmonary cases show more or less serious aryngeal damage.

Temporary hoarseness was noted in 1339 out of 3007 cases seen at he Phipps Institute while persistent hoarseness was noted in 520 out of 466 cases. Fetterolf in a clinical post-mortem study of the larynx in 00 cases dying in the Phipps Institute found that it was tuberculous in 3; non-tuberculous in 13; and doubtful in 4.

Dysphagia.—Difficulty in swallowing is not common in tuberculous atients except with involvement of the pharynx or larynx, usually the atter. Involvement of the epiglottis is especially liable to interfere with wallowing because of pain which may be so intense as to prevent even he swallowing of water. In some instances difficulty in swallowing is lue to interference with the nervous mechanism governing the larynx. This, however, is quite rare.

Dyspnea.—Shortness of breath is a frequent complaint among those uffering from tuberculosis. Curiously enough the degree of dyspnea ears very little relation to the amount of pulmonary damage. Little r no inconvenience is experienced by some people, except on exertion, n spite of extensive disease while in others, with relatively little trouble, his symptom is very troublesome. While it is undoubtedly true that he curtailment of the breathing space has some influence on causing hortness of breath there are other factors which seem to exert a more narked influence. Shortness of breath is more apt to be present if there s fever and is apparently dependent also on the nervous condition of the ndividual. Patients who are nervous and apprehensive about their ondition are more apt to suffer from shortness of breath and tachycardia han those of a phlegmatic temperament. Immobility of the diaphragm s a common cause of the shortness of breath. Some degree of shortness of breath was noted in 80 per cent. of 3007 cases at the Phipps Institute. As a rule the shortness of breath becomes noticeable only on exertion, uch as, going upstairs, climbing a hill, or walking fast.

Physical Signs.—Before taking up in detail the physical signs it may be well to emphasize the importance of keeping in mind the morbid natomy, and the symptomatology of tuberculosis in their relation to he physical findings.

The three factors involved in the art of physical diagnosis are so lependent on one another that it is not possible to say that one is more mportant than the other. A knowledge of all three is essential. It is necessary, for instance, to know what portion of the lung is first involved, he character of the pathological process and how it advances or retrogresses, as the case may be. Knowledge of this sort and its application o physical signs is to be learned largely in the deadhouse, and not at the edside. Austin Flint expressed the importance of this association very clearly when he stated that: "The significance of signs which rep sent abnormal physical conditions rests on the uniformity of their assoc tion with the latter—certain physical signs denote certain abnormal con tions, because clinical experience, inclusive of the study of lesions wi the scalpel, has sufficiently established the fact." And again he stat in regard to physical signs that: "Invaluable as they are, their importar is greatly enhanced by association with symptoms and the knowledge pathological laws. The results of physical exploration alone frequent leave room for doubt and liability to error, when a due appreciation vital phenomena and of facts embraced in the natural history of d eases insures accuracy and positiveness. An overweening confidence the former is to be deprecated, as well as exclusive reliance on the latt And, since the practical discrimination of intrathoracic affections always to be based on the combined evidence afforded by these thr sources of information, in treating of the subject it is desirable that t attention shall not be limited to one source to the exclusion of the others

We are largely indebted to the French pathologists for pointing o the necessity of comparison of all symptoms of pulmonary disease a of connecting this comparison with their succession in order. This pha of the subject was especially dealt with by Andral, and more particula by Louis in the promulgation of his numerical theory.

Gerhard also emphasized the great importance of the comparison the general symptoms and physical signs. As he points out, the earl writers on physical diagnosis, especially Laennec, were rather dispos to separate physical from symptomatic diagnosis. And, although tl error depended on the novelty of the art and the overstrained efforts extend its application, it still persists, although possibly to a less exte than formerly. Not only are we to avoid exclusive reliance on the pr ence or absence of physical signs, but it is also to be borne in mind tl physical signs, while often indicating the extent and degree of pulmona damage, convey no direct information as to the pathological nature those changes.

As to whether one shall take a radical or a conservative view in ca of a doubtful nature, there does not seem to be, in the writer's opini any room for argument. A doubtful case is at best still doubtful, a such being the case, one should exercise every precaution to exclude presence of tuberculosis, always bearing in mind that the disease in very earliest stages is, as a rule, extremely amenable to treatment. the other hand, the more marked the physical signs, the greater damage to the lung and the more uncertainty as to whether the dise can be arrested.

Inspection.—This method of physical examination is too frequen omitted, or made so hastily and cursorily that little or no informat is obtained. As a matter of fact, inspection properly done yields m valuable information than any other procedure at our disposal, next auscultation. And furthermore, it has this to commend it, namely, tl no special training is required and the beginner, providing he uses eyes, is as capable of seeing defects as the trained observer. This is marked contrast to the training necessary to educate the ear to differ tiate sounds, particularly those produced by percussion, the latter of taking years of practice. One who has been taught to make a pro inspection can in the majority of cases of tuberculosis, from this meth alone, determine the side affected, and approximately the extent of the lesion. As this method takes no special training, it is especially valuable to the student and to those who see chest cases incidentally, and not constantly.

In order that inspection should yield the best results it is absolutely essential that the patient be stripped to the waist, and so placed that the parts under inspection are equally exposed to the light, as an error may occur if one-half of the chest is less well lighted than its fellow.

Before directing special attention to the chest itself it is important to look for abnormalities in other regions, although the earlier the disease, the less frequently do we find anything of moment. In a large number of cases the pupils are unequal in size, the dilated pupil being on the affected side. Flushing of the cheek on the affected side may be present as an early manifestation, but is more frequently encountered later. The mouth should be examined at this time, as a matter of routine, and any abnormalities of the teeth, tonsils or upper respiratory tract noted. Information of this sort, however, is of value from the standpoint of treatment rather than diagnosis.

The *thyroid gland* is often enlarged, although this may be more apparent than real owing to emaciation. Among 2122 patients seen at the Phipps Institute enlargement of the thyroid gland was noted in 135 (6.69 per cent.). In many of these cases the eye phenomena, so commonly seen in exophthalmic goitre, are also present. Whether hyperthyroidism exists or not in these cases can be determined by the injection of 7½ minims of adrenalin. If such is the case the blood pressure is raised and the phenomena usually associated with this condition are accentuated, if slightly present, or are brought to light if unsuspected. The test is of service in determining whether extensive constitutional symptoms are due in part or entirely to hypersecretion of the thyroid gland.

Inspection of the *hands* often gives most valuable information. The nails especially should be carefully examined. The most common change encountered in the nails is a tendency to curving, without any associated clubbing. The nails not only curve over the end of the finger slightly, but they are narrower from side to side than normally. The color may be normal, or it may be an exaggeration of the normal, whitish-pink color. While violet or bluish-colored nails are of frequent occurrence in the advanced stages of the disease, they are not usual in the early stages; when present at this time they are extremely ominous. Their presence, in the early stages of tuberculosis, should cause one to be very guarded in predicting the outcome, no matter how slight the involvement may seem from the physical signs, as they usually indicate an especially severe form of infection.

Clubbing of the fingers, although usually stated to be commonly associated with pulmonary tuberculosis, is as a matter of fact, not frequent, and even when present, not marked. The extreme grades of clubbing of the fingers occur for the most part in non-tuberculous affections, notably empyema, bronchiectasis and congenital heart disease.

In the early stages the *skin* may present a mottled appearance indicative of a poor vasomotor tone. Profuse sweating either at night or after exertion is not common at the onset of the disease, but axillary sweating, often noted during the examination, is sufficiently frequent in these cases to be worthy of note.

Taking up the chest proper, it is usually best to note, when visible, the position of the apex beat of the heart, as in addition to disease of the heart itself, the apex may be displaced out of its normal position as the result of disease of the lungs or pleura. An effusion displaces the heart toward the opposite side while fibroid changes in the lung draw the lung toward the affected side.

Coming now to inspection of the *chest* with a view of detecting pleural or pulmonary mischief, it must be borne in mind that we are dealing with comparisons. In regard to the shape of the chest, no information of importance is obtained in the incipient stage. The majority of patients at the onset of the disease present well-shaped chests, which do not present any marked abnormality. Thus Pope and Brown found 83 per cent. of well-formed chests in 193 incipient cases. The so-called paralytic thorax, which is sometimes seen, is not uncommonly noted in individuals with a marked hereditary taint and antedates the tuberculosis rather than being caused by it. The paralytic thorax is long and narrow and apparently flattened in the antero-posterior diameter, the clavicles and scapulæ are prominent, the latter being tilted outward from the chest and away from the spine. The ribs are oblique, forming an acute angle with the sternum. (See Figs. 13, 24, 31, 35.)

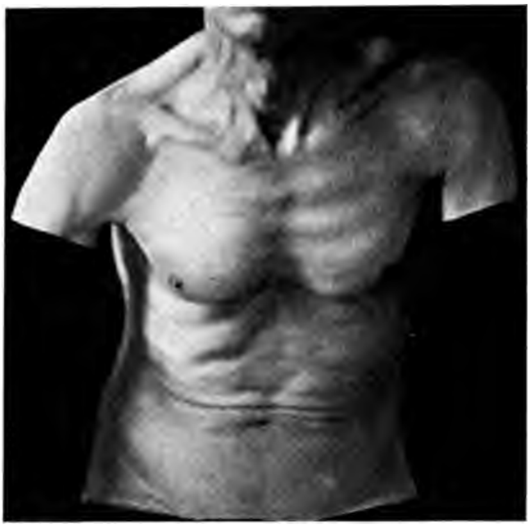

FIG. 251.—Shows drooping of the right shoulder and the long line from the neck to the point of the shoulder.

The following points should be noted: (*a*) Whether the shoulders are on a level; a very early sign is drooping of the shoulder on the affected side. In this connection, however, it must be recalled that certain occupations, such as clerks, predispose to a faulty sitting posture, which will raise or lower one shoulder.

(*b*) The line from the neck to the point of the shoulder. This line is normally slightly convex, but with beginning disease at one apex, it tends to become straight and also longer than its fellow, the latter defect being brought about by the drooping of the shoulder (Fig. 251).

(c) The degree of prominence of the clavicles. In most well-nourished nen the clavicles are scarcely, if at all, visible; in men, on the other .d, unless unusually fat, the clavicles are more or less prominent. Associated with undue prominence of the clavicle is an exaggeration he supraclavicular fossa. A noticeable amount of flattening may be ed also beneath the clavicle.

(d) *Expansion.*—Deficient expansion of the chest wall overlying the x of the lung is one of the most valuable signs of early tuberculosis ich we possess. It is especially to be looked for toward the outer der of the lung, just beneath the clavicle. In this situation the st wall normally balloons out quite markedly. If, however, there its an infiltration of tubercles at or near the apex, the underlying lung s not expand as fully as its fellow, or if the expansion is equal on both s the affected side tends to lag behind slightly, especially at the inning of the inspiratory period.

Inspection of the apices for the purpose of determining the degree expansion yields the best results, if but one apex is involved. If re exists disease at the summit of both lungs the value of comparison hus lost, and one may be unable to come to any definite conclusion n this method alone. Two other methods of determining slight ounts of retraction on the affected side are available, namely, palpation mensuration, both of which will be described in detail later.

Inspection of the chest posteriorly. In the early stages of tubercu- s the amount of information gained from the posterior view of the st is slight compared to what can be learned from the anterior view. ery common occurrence in tuberculous subjects is the presence of venules in the region of the nape of the neck. These small veins be bluish or purplish in color. They have been cited as an evidence the existence of pulmonary tuberculosis, but are so frequently ountered as to be of little value as a diagnostic sign.

Owing to the interposition of the scapular muscles and the ribs little be noted as to expansion in the upper part of the chest. At the bases, ever, expansion can be determined in the same manner as over the es anteriorly. The most noticeable finding is the presence of varying rees of atrophy of the muscles in the supraspinous fossa. If the arms allowed to hang naturally at the sides, the angle of the scapula on the cted side may tip backward slightly more than its fellow. This lency to "winged scapulæ" becomes much more marked as the ase progresses.

Slight degrees of scoliosis may be present. Litten's sign may be ent (see Part I, p. 29). The sign is not often employed as there is ther and easier method of determining the mobility of the lower part he lung, namely, by percussing the lower border of the lung during ed expiration and inspiration. The fluoroscope may be employed
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Palpation.—Tactile fremitus is a sign of comparatively little value in oient tuberculosis, as the amount of infiltration in the underlying is usually too slight to produce much exaggeration over the normal. normal discrepancy between the two apices has already been alluded ee p. 74).

Pottenger has called attention to *rigidity of the muscles* over the ted area. Recently Galecki[1] reports on finding this sign present in

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[1] *Beiträge Zur Klin. d. Tuberculose,* 1914, xxx, No. 3.

93 per cent. of recent cases and not at all in cases with a healed lesion. The sign is elicited by light touch palpation. This sign is not to be confused with myoidema. The latter term is applied to a local contraction of the muscle, produced by direct percussion, and causing a nodular swelling, which arises immediately after percussion, lasts a second or two, and then gradually disappears. It may be produced two or three times and then cease to appear. It is best seen in the pectoralis major muscle. Although this phenomenon is commonly encountered in tuberculosis, it is not peculiar to the disease.

Palpation, however, is an invaluable method, for determining the *amount of expansion* at the apices in those instances where the difference between the two sides is slight and one is in doubt from inspection alone. In determining the degree of expansion over the apices anteriorly by means of palpation, one of two procedures may be followed. The examiner sits squarely in front of the patient and places one hand in the same relative position beneath each clavicle; he should then close his eyes, or turn his head aside. In this way even the very slightest variation may be noted. Or the examiner can watch his two hands and determine which moves the most. The former is by far the more delicate method. The value of the sign is enhanced by the readiness with which it is elicited. Students with but a rudimentary knowledge of physical diagnosis can readily detect a slight difference between the two apices when the other signs, indicative of a lesion, are too vague to be appreciated by an untrained observer. In the incipient stage palpation is not apt to reveal abnormalities in portions of the chest other than one or the other apex.

Mensuration is the least used of the various procedures of physical diagnosis. At one time a great deal of stress was laid on the degree of expansion of the chest, good expansive power being looked upon as indicating freedom from thoracic disease; insurance companies still insist on a record of the difference between expiration and deep inspiration. In the absence of more convincing signs it is doubtful whether any importance can be attached to a degree of expansion below the normal (approximately $2\frac{1}{2}$ inches), if this is the only evidence obtainable.

A more useful method of employing mensuration is by means of the lead tape *cyrtometer*. This method is too little used. While it is valuable for diagnostic purposes, its greatest usefulness is in depicting the changes in the contour of the chest as the disease progresses, either to a favorable or an unfavorable termination. The technique of the method is readily acquired with a little practice. The lead cyrtometer consists of a piece of sheet lead, $\frac{3}{16}$ inch thick, $\frac{1}{2}$ inch wide and 26 inches long. It should be covered with thin calfskin. In addition there is required a pair of obstetrical calipers capable of opening at least 12 inches. The first step is to obtain the antero-posterior diameter of the chest. Minor recommends for the two fixed points, the middle of the sternum at the level of the fourth costal cartilage in front, and the eighth dorsal spine posteriorly. The latter is a little below the level of the inferior angles of the scapulæ. Having, with the calipers, ascertained the depth of the chest between the above-mentioned points the distance is marked on a sheet of paper, capable of receiving the tracing of a chest 12 inches in its antero-posterior diameter and 16 inches in its lateral diameter.

Each half of the chest is taken separately. With the eighth dorsal e as the fixed point, one end of the tape is firmly held so that it will slip and is then brought around to the anterior fixed point. The tape ld be firmly applied so that it fits snugly. In crossing the axillary ce care must be taken to mould the tape to the chest wall, otherwise space is apt to be bridged. The anterior point can be marked by

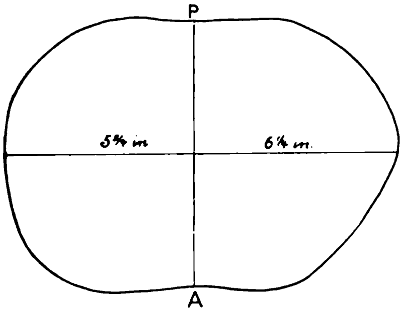


FIG. 252.—Slight retraction of right side. Lesion at right apex

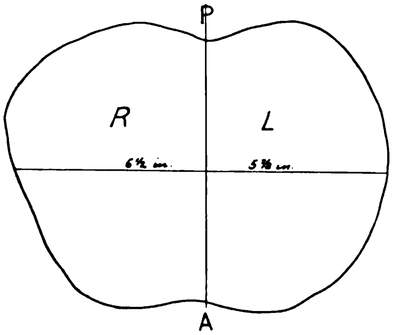


FIG. 253.—Slight retraction of left side. Lesion at left apex.

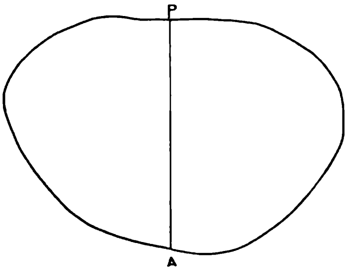


FIG. 254.—Advanced bilateral disease. Marked retraction of right side.

enting the leather with the finger nail. The tape is then carefully oved and the two ends placed over the marks indicated on the paper the calipers. By means of a pencil the perimeter is then traced on the er. The left side is similarly taken (Figs. 252, 253 and 254).

By using different colored pencils at each subsequent tracing, one ains an excellent picture of the contour of the chest. Even in very early cases there is a slight amount of shrinkage on the affected side, and the greater the amount of disease present the greater, as a rule, is the degree of retraction. As the case progresses toward recovery the affected side tends to fill out so that eventually discrepancies between the two sides disappear. In some early cases with a marked degree of shrinkage, the reëxpansion is quite rapid. On the other hand, if the disease advances, the affected side shows an increased amount of retraction, and with involvement of the sound side evidences of shrinkage will likewise appear. Minor states that the increase of the perimeter takes place on the unaffected side first as a result of the compensatory action of the sound lung, and that the increase of the affected side generally follows the increase of the unaffected side. The increase can be in breadth or depth; the latter is of more significance, however, as the tracing of the former may be affected by an increase in the amount of fat and muscle, while the latter being measured between two bony points is not so affected. Thus it can be seen that the method is a valuable diagnostic, as well as a prognostic aid. One precaution should be kept in mind, namely, as to whether the individual is right-handed or left-handed.

Percussion.—Before undertaking to describe the percussion changes in early tuberculosis, it is necessary to emphasize several important facts.

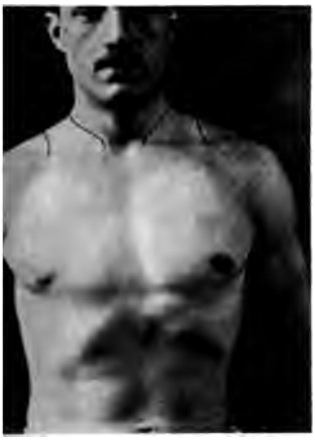
1. There is a normal discrepancy between the percussion notes of the two apices. This has been recognized for many years, but advantage is not always taken of the knowledge. The note on the right side is normally a little higher in pitch and a little less resonant than the note on the left side. Flint described the note on the right side as vesiculotympanitic without, however, advancing any reason for the change. Recently Fetterolf and Norris have given a satisfactory explanation of the difference. Their study, from both the clinical and anatomical standpoints, shows quite clearly that the right apex is smaller than the left (see Figs. 62, 63, 64, 76 and 77) and that furthermore the position of the blood-vessels on the right side tend to diminish the resonance. The close approximation of the right apex to the trachea (see Figs. 49, 75, 95 and 104), the latter giving a tympanitic note, thus tends to raise the pitch of the percussion note; the left apex, being larger and having large blood-vessels and areolar tissue interposed between it and the trachea, gives a pure resonant note.

2. One of the difficulties the beginner has in percussing the apices is that to his ear the note is frequently impaired. The real difficulty, however, is that the note is less intense in this region owing to the small amount of lung tissue at the apex as compared to the base, and also because of the distance of the lung from the surface over which the percussion is being applied. These differences apply to the posterior aspect of the apex, and to a less extent, the area above the clavicle. Anteriorly beneath the clavicle the pulmonary tissue lies immediately beneath the chest wall so that the note is usually intense, and on the left side typically resonant (see Figs. 260, 261, and 262).

3. Keeping in mind this normal difference, it must be remembered again that we are dealing with comparisons, and inasmuch as the changes are at best slight, each side must be compared carefully with the other. If slight changes exist at the summit of both lungs it is probable that very little definite information will be forthcoming from percussion.

Percussion of the apices in a case of suspected incipient tuberculosis dure that requires a well-trained ear, and not a little experience. ge from the normal is usually so slight that for the beginner the s the least fruitful of results; and even the experienced observer ubtful cases, apt to be influenced in his interpretation by the or absence of symptoms, or other associated physical signs. ction of slight changes at the apex is facilitated by marking with ncil the borders of what is known as "*Krönig's isthmus.*" This d of resonance which crosses the shoulder (Figs. 255 and 256). owest point is at the top of the shoulder, and in both front it widens out to meet the extended areas of resonance beneath cle and supraspinous fossa. The value of this sign arises because ell-known tendency of the lung with a developing tuberculous shrink, either as the result of fibrosis, or of lessened functional

apping out the "isthmus" it is well to begin the percussion well ide of the neck and gradually come downward until a change


G. 255.—Krönig's isthmus.
Normal anterior view

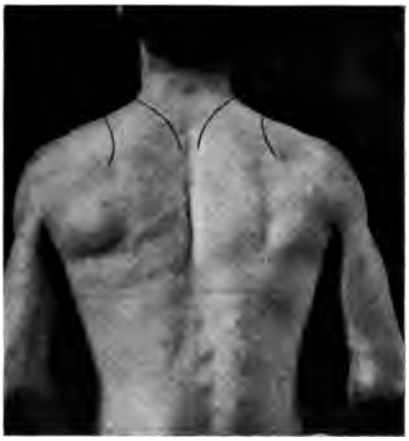


FIG. 256.—Krönig's isthmus.
Normal posterior view.

n-resonance to resonance is noted. This point is marked with cil and by working either forward or backward the inner line is ut. The outer line is mapped out similarly by approaching the t area from the point of the shoulder. The inner line, except at er anterior extremity, is concave and runs downward and for- ding just a little outside the sterno-clavicular joint. Posteriorly r line inclines toward the spinal column; at the level of the second ertebra it continues parallel with the spinal column, at a distance t ½ inch.

outer line, anteriorly, runs downward and outward, ending nction of the outer and middle third of the clavicle. Posteriorly ownward to about the middle of the spine of the scapula.

value of this procedure lies in the fact that while one may be in s to the quality of the note, if percussion is made directly over ation of normal resonance, one is less likely to err if the normal approached from non-resonant parts, such as the neck or shoul- ne quickly learns to appreciate what the normal width of the isthmus should be, and if this becomes narrower it is an indi trouble in the underlying apex. If but one side is diseased, the side will show a much narrower "isthmus" than the healthy s 257 and 258). As the disease becomes more extensive at the two lines of the "isthmus" tend to become closer and clc finally in the advanced case no semblance of resonance remain

Some observers have laid stress on direct percussion of the without the intervention of a pleximeter, the claim being made tha a small area of impairment can be detected that would otherw detection. Such instances may occur, but they are far fr common.

Having outlined the apices the percussion should be contin ward until the base of the lung is reached. Even in incipien

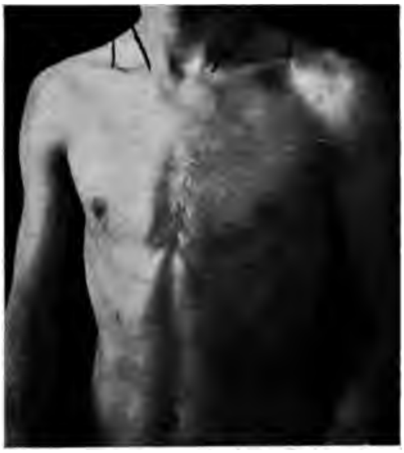


Fig. 257.—Krönig's isthmus. Normal on left side. Narrowed on right side due to tuberculosis of right apex.

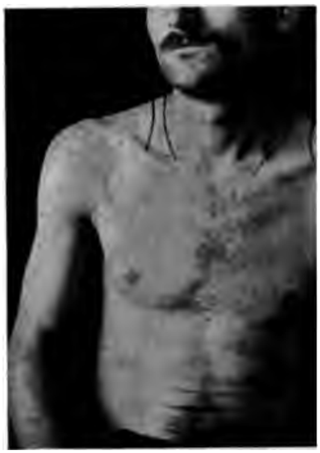


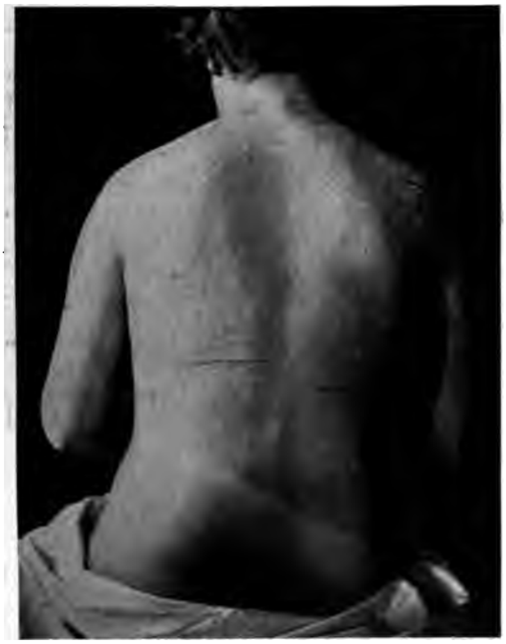
Fig. 258.—Krönig's isthmus. narrow due to bilateral tub

will usually be found that the resonance does not extend quite the affected as on the unaffected side after deep inspiration (see It will be recalled that mensuration shows some diminution i of the affected side. Furthermore, it has been shown by flu examinations that the descent of the diaphragm on the affect usually diminished. This is known as *Williams' early diap sign* (see p. 646). These observations indicate that the lung, e the seat of a small amount of disease, functionates less freely unaffected lung, or that the unaffected lung is functionating n the diseased one. Whichever is the correct explanation, the fac that the resonant note is apt to stop at a higher level on the than the unaffected side.

With ordinary quiet breathing the bases of the lungs exte level of the tenth dorsal vertebra; the complementary space of t cavity, however, extends to the level of the twelfth dorsal On deep inspiration the lung can be made to expand for an inc below the level of the tenth dorsal vertebra, providing it or t m disease. If, however, the lung is much diseased, or the rity is obliterated, or the diaphragm is immobile, the base affected side remains stationary.

marked out the borders of the lung, the heart and viscera ship to the lungs should be outlined.

ation.—The fact that there normally exists a difference be- right and left apex has already been alluded to (see p. 74). urther need be said except to emphasize the importance of is in mind. One other fact should be mentioned, namely, the portance of the different steps taken to determine whether one er apex is the seat of tuberculous disease. That auscultation t important means at our disposal for the detection of intra- sease, there can be no doubt. On the other hand, it is equally



259.—Restriction of motion at base of left lung. Lesion at left apex.

n those instances in which the pulmonary damage is slight aus- lone, valuable as it is, will frequently fail. *The recognition of ent tuberculosis cannot be accomplished except by a careful con- of the facts revealed in the history, and a proper estimation of eviation from normal as revealed by inspection, palpation, men- ıd percussion.* Even a skilled auscultator would often be in) the presence of a slight tuberculous deposit if he relied on aus- lone. The question as to whether suspicious breath sounds nsidered normal or abnormal not infrequently hinges on the of the information obtained in the history and by the other f physical exploration. This digression has seemed necessary because of the absolute reliance so many physicians place on auscultation alone.

Granular Breathing.—This type of breathing, which owes its importance as an early diagnostic sign to Grancher, and in this country to Minor, is now regarded as the earliest manifestation of the auscultatory changes in pulmonary tuberculosis. While readily recognized after one has heard it a few times, it is a sound not easily described by words. Granular breathing is a rough or sputtering type of breathing. Turban has likened this type of breathing to the rapid succession of minute explosions; Minor to "a succession of very short sounds, as though small, soft granules of fine, wet sago were being rolled over each other." Perhaps the clearest description is that it suggests the coexistence of râles, and yet, just as the listener fully expects to hear fine râles at the end of inspiration, the inspiratory phase ceases. This type of breathing has been described as being due to slight narrowing or uneven surface of the bronchioles, or to a rapid interruption of the air entering the alveoli about the tuberculous deposit. The following seems to us a more plausible explanation. It should be recalled that in the early stages of pulmonary tuberculosis there is a considerable amount of relaxation and collapse, or partial collapse of the vesicles immediately around the tubercles. As the air forces its way into these partially collapsed vesicles they expand independently instead of synchronously. This imparts to the inspiratory murmur a jerky sound and also gives the impression of crepitation due to the separation of the slightly moistened walls of the air vesicles.

Feeble Breathing.—Next in importance to granular breathing is slight enfeeblement of the respiratory murmur. This type needs no special description. If on comparing the two apices the breath sounds are less intense on one side than the other, the fact is significant. It is usually taught that enfeebled breathing to be of significance as an early sign in tuberculosis must be limited to the apex. It has been our experience, however, that the breath sounds all over the affected lung, even with very slight apical signs, are not infrequently less intense than over the affected side. This is after all not surprising, when we recall that mensuration shows a diminution of the affected side and the fluoroscope a heightened diaphragm.

Prolonged Expiration.—Prolonged expiration, although not the earliest change from the normal in the breath sounds, is the most usual finding, as the two earlier changes described above often escape detection. The respiratory murmur in this type of breathing may be harsh or slightly suppressed, but in either instance the characteristic feature is the prolonged, high-pitched, bronchial quality of the expiration. Heard at the left apex, one is rarely in doubt as to its significance; when confined to the right side, there is apt to be a certain amount of question as to whether we are dealing with normal or pathological broncho-vesicular breathing. While in every normal chest there is more or less marked broncho-vesicular breathing at the right apex, there is no definite standard and the question of whether it is pathological or not is usually settled by the presence or absence of collateral evidence. In children especially there is a strong tendency towards exaggeration of the normal signs at the right apex, and not infrequently children are said to be tuberculous, because of the strong transmission of both the spoken and whispered voice and the prolonged blowing character of expiration.

Cog-wheel or Wavy Breathing.—Cog-wheel or wavy breathing has been described as an evidence of incipient tuberculosis, but the best authorities now are agreed that it can no longer be considered of importance as an early sign. As the name indicates it is an interrupted type of breathing. The inspiratory phase is the one commonly subject to the interruptions; rarely the expiratory. It may occur in a patient suffering from the pain of acute pleurisy, or in nervous or chilly individuals. In tuberculous subjects it is usually heard over areas which divide healthy from diseased tissue. Cog-wheel breathing is not to be confounded with the cardio-inspiratory murmur of which we will speak presently.

Vocal Resonance.—The alterations in the voice sounds are not of great value in early tuberculosis, as the deviation from the normal may not be sufficient to be appreciated. The whispered voice is normally heard with distinctness over the second costal cartilage on the right side, and posteriorly in the interscapular regions. In many chests it is indistinctly heard also over the extreme right apex anteriorly and posteriorly. Indistinct whispering pectoriloquy in the latter situation is of no significance without collateral evidence. As an indication of infiltration the whispered voice is subject to the same rule as the spoken voice.

Râles.—I have already pointed out that in the earliest change of the breath sounds (that is the "granular breathing") one gets the impression that râles are about to be heard, but as a matter of fact are not. In true incipient tuberculosis, no matter what the type of breathing may be, râles are not heard with ordinary quiet breathing. If, however, a deep inspiration is taken, fine, dry crackles may be elicited above or just below the clavicle, or above the spine of the scapula, posteriorly on the affected side.

An invaluable procedure for bringing out these râles is after auscultating the chest, while the patient is breathing quietly through the mouth, to have him give a short cough, followed by a moderately deep inspiration. This is repeated first on one side, then the other until the entire chest has been gone over. In this way small, localized areas containing these fine râles are detected which would otherwise escape observation. If crackles are confined to an apex and do not disappear after deep breathing or coughing, they are the strongest kind of evidence of the existence of a pulmonary tuberculosis. In other situations they are of less importance as a diagnostic sign of tuberculosis.

Râles that are heard with ordinary quiet breathing are somewhat coarser, and in addition give the impression of moisture. Râles heard under these circumstances are not evidence of incipiency, but indicate that the disease has probably passed that stage. This is true also of the arger moist râles (the so-called mucous click, etc.) even though limited o one apex.

Cardiac Phenomena Occurring in Tuberculosis.—Most of the abnormal gns of cardiac origin in tuberculosis manifest themselves in the second nd third stages of the disease.

The heart sounds are at times unduly transmitted toward the affected pex, even in the incipient stage, and the denser the infiltration the more tense do the heart sounds become.

Cardio-respiratory murmurs are common in tuberculous subjects, and hile they have lost much of the diagnostic significance attached to them the older clinicians, it is surprising how frequently they are encountered in tuberculous patients. They are as a rule associated with a rapid cardiac action (see p. 254).

While the cardio-respiratory murmur may be heard in the incipient stage, it is more commonly encountered in the moderately advanced stages of the disease. It is also encountered in individuals with an old healed lesion at one or the other apex. In the same category may be placed *systolic murmurs* heard in the *subclavian arteries.*

The frequency of the heart's action is not greatly accelerated in the early stages; if so, it is an indication of marked toxemia. A single office observation as to the frequency of the heart's action is untrustworthy because of the nervousness and excitement often incident to the examination. To be of value the pulse rate must be observed with the patient at rest for a number of days. As the disease progresses the heart's action almost invariably increases in frequency. With the development of tachycardia, even moderately severe, reduplication of the heart sounds is frequent, particularly the second pulmonic; less frequently the first sound at the apex. With extensive disease of the left lung, retraction of its anterior border is common. This results in visible pulsation of the heart in the region of the second costal cartilage on the left side.

At one time it was currently believed that an individual who suffered from *organic heart disease* was not apt to become tuberculous. As Norris pointed out some years ago, there is no support for this theory, and one is apt to encounter an associated organic lesion of the heart as frequently in tuberculosis as in any other disease. In addition to organic murmurs, accidental murmurs of unknown origin are frequent, particularly in the latter stages of the disease. Functional murmurs, systolic in time, are of not infrequent occurrence at the apex, but by far the most frequent murmur of this type is a systolic murmur heard at the base of the heart, at or near the second pulmonic area.

Physical Signs in the Second or Moderately Advanced Stage.—*Inspection.*—When the disease has progressed sufficiently to be designated moderately advanced, the general health of the patient begins to show impairment (see Fig. 230). The loss of weight is apt to be noticeable and the muscles present a flabby condition. Slight flushing of the cheek on the affected side is frequently noted and this is in striking contrast to the anemia.

Skin eruptions which are not uncommon in the third or advanced stage may make their appearance during the second stage. The commonest of these eruptions is pityriasis versicolor caused by the *microsporon furfur.* ?

As to the chest itself the affected side shows more marked evidence of shrinkage. The clavicle is more prominent; the fossa above the clavicle is deeper and flattening beneath the clavicle more noticeable. Diminution of expansion which, in the incipient stage, is not always apparent on inspection, becomes readily so in this stage.

Some atrophy of the muscles overlying the affected apex is apt to make its appearance at this time. Including all types of cases atrophy of the muscles was noted at the Phipps Institute in 1325 cases out of 4343 —931 on the right side and 394 on the left. In women the breast on the affected side may be distinctly smaller than that on the opposite side.

Varying degrees of spinal curvature are also of frequent occurrence and the more extensive the disease the more marked does this tendency

e. Spinal curvature was recorded as having been present in 968 3436 cases of all types at the Phipps Institute.

heart is not apt to be noticeably displaced at this time.

pation.—This confirms the diminished respiratory movement e affected area already noted on inspection. The tactile fremitus lly exaggerated but may show no appreciable change.

cussion.—While in the incipient stage one is often in doubt as to r the percussion note deviates from the normal; this is rarely so he infiltration has become more dense and more tissue is involved. majority of instances the percussion note is not entirely devoid of ice because the tuberculous infiltration is rarely massive. Patches earing tissue still exist about the tubercles and for this reason ; degrees of pulmonary resonance persist. In some cases a tym- note is obtained as the result of cavity formation. This, however, ly a manifestation of advanced disease, the signs of which will be red in detail later.

nig's isthmus becomes narrower in the second stage and the im- ercussion note may extend as low as the third or fourth rib (see 3). By the time the infiltration has become marked at the apex inkage of the affected side is apparent the percussion note at the l be found to be on a higher level than the sound side. With the advanced to the second stage at one apex there may be signs ve of the first stage in the opposite apex.

suration.—If a cyrtometer tracing has been made during the first second tracing made after the disease has advanced to the second ows more shrinkage and if, in the meantime, the opposite apex ome involved, that side will also show some diminution in size.

cultation.—The breath sounds in the second stage of the disease a rule, sharply differentiated from those heard over the healthy d from those heard in the first or incipient stage. The granular, tly enfeebled or doubtful broncho-vesicular breathing becomes ly broncho-vesicular; the latter may be slightly suppressed or may approach true bronchial breathing. Bronchial breathing such rd in croupous pneumonia, when the lung is completely solidified, ommon in tuberculosis. This is because the infiltration rarely s massive, as a rule remnants of healthy tissue remain between the areas and thus impart a vesicular quality to the respiratory r. The broncho-vesicular breathing may therefore be slightly al in character or very markedly so, depending on the density of ltration. When the infiltration has destroyed most of the pul- tissue in a given area, the chief characteristic of the breath sounds they are greatly suppressed. At times they may be almost le, especially if râles are present.

le in a few cases broncho-vesicular breathing may gradually pass pure bronchial type it far more frequently happens that caver- amphoric breathing appears as the result of cavity formation. lower border of the infiltrated area, cog-wheel breathing is tly heard.

al resonance is a sign of relatively little value. It is usually ex- ed and the increase corresponds closely to the percussion changes.

s.—As there is usually an associated bronchitis and some soften- he caseous areas medium-sized râles are common. They may be very numerous or one may hear only a few isolated râles; the latter may be constantly present or may be heard only during every second or third inspiration or expiration. Very often when but one râle is heard it has a peculiar sticky quality; it is often referred to as a mucous click. By some it is considered almost pathognomonic of tuberculous infiltration. While not absolute, one is safe in saying that the more numerous the râles the greater is the probability of softening and hence the more serious the outlook. If the râles have a metallic quality, a cavity may be suspected even if other signs of excavation are absent. Some crepitating râles are commonly present in this stage but in some instances they are not heard while the patient is breathing naturally. Having the patient cough and then take a deep breath, or breath out and then cough, will often bring out a shower of fine râles; sometimes deep breathing will accomplish the same result.

Accordingly as the râles diminish or increase or change in character one is often able to foretell the probable outcome of the disease. If the râles diminish it is an indication that the associated bronchitis is disappearing and that the softening of the caseous areas is ceasing. On the other hand, if the râles gradually become more numerous breaking down of the lung tissue is probably taking place.

With arrest of the disease the râles, particularly the medium-sized ones, may entirely disappear. The crepitating râles, on the other hand, may persist for years even when the patient is entirely free from symptoms.

Pleuritic friction sounds are frequently heard during this stage. The friction rub may occur on the affected side or it may be heard on the opposite side. It is often difficult to distinguish between crepitating râles located in the lung and fine pleural crepitations. At times these fine crepitations may be heard over both the upper and the lower portion of one lung. While it is not always possible from the character of the sound alone, to differentiate them, one can usually judge whether the râles are entirely due to infiltration of the pulmonary tissue or whether those heard over the lower portion of the lung are due to pleurisy, by the character of the symptoms. If the entire lung is infiltrated the patient is almost certain to have serious symptoms; on the other hand, if the symptoms are mild and the patient in good condition the lower portion of the lung is presumably free from disease and the râles are pleuritic.

The Heart.—In many instances the heart presents no abnormalities. On the other hand, there may be noted in this stage an increase in the number of heart beats per minute. A cardio-respiratory murmur is quite frequently encountered.

The nearer the disease approaches the third or advanced stage the more frequently does one hear abnormal heart sounds. The first sound may be reduplicated and reduplication and accentuation of the second pulmonic sound are commonly heard. Accidental murmurs may also be heard particularly at the pulmonic area. As a rule the systolic blood-pressure is low.

Physical Signs in the Third or Advanced Stage.—In taking up a consideration of the physical signs encountered in the advanced stage of pulmonary tuberculosis the task is at once very easy and at the same time very difficult. It is easy because inspection alone will frequently indicate that serious mischief has been wrought by the disease; it is often difficult, however, to indicate the exact nature of the damage because of the extremely varied pathological changes which are present. In one and the same lung there may be cavity formation, consolidation and varying degrees of infiltration; and in addition there may be present an effusion, a partial pneumothorax, or involvement of the pleura, which further complicates the picture.

With reasonable care in the examination of the chest the examiner should be able to determine the approximate amount of damage present. The more experienced the physical diagnostician the more clearly will the physical signs he elicits correspond to the actual pathological changes.

Inspection.—When the disease has progressed to this stage the vast majority of individuals present the appearance so familiar to all. The most dominant feature of the picture is the extreme emaciation, a fact that gave the disease its first name, phthisis or the wasting disease. The hair presents a lanky, lusterless appearance, the temples and cheeks are sunken and the eyes look unnaturally bright and feverish. The so-called hectic flush is usually present and is more marked on the side most diseased; the bright color of the cheeks is in marked contrast to the blanched, waxy appearance of the surrounding skin. Flushing of the cheeks usually occurs synchronously with the afternoon rise in the temperature but may be noted before the thermometer shows the presence of fever.

The skin is usually dry and very often scaly. Pityriasis versicolor is frequently noted, especially among those who are not cleanly in their habits.

Inspection of the mouth at this time may show a tuberculous ulceration of the tongue, the buccal surface of the cheeks, the tonsils or the pharynx.

The hands in many cases are noticeably altered. In the vast majority of advanced cases the finger nails present a slightly bluish color. The nails themselves are often curved, usually from side to side but they may also curve over the end of the finger. Clubbing of the fingers is not so frequently encountered in tuberculosis as usually is taught and when present is rarely of the extreme grade seen in bronchiectasis, congenital heart disease, or empyema. Clubbing of the fingers and extreme curving of the nails, when at all marked in pulmonary tuberculosis, are usually evidences that the disease has been of long standing and that there is considerable fibrosis and bronchial dilatation present in the lungs. Clubbing of the fingers was noted at the Phipps Institute in 21.7 per cent., and curving of the nails in 38.9 per cent. of 3551 cases.

In the terminal stages of the disease edema of the lower extremities is not infrequent. A small percentage of cases towards the end show a purpuric eruption usually over the legs and thighs.

Inasmuch as individuals with any type of chest may acquire the disease the only striking features in many instances are the emaciation, the
rspaces and evidences of retraction on one side or the other.
on will usually show marked flattening at both apices and
nd lack of motion at one base. As a rule the motion is good
of the lung least diseased.

t.—More or less displacement of the heart is extremely com-
esent in about two-thirds of advanced cases. This often
d by inspection but in many instances the position of the

heart can be determined only after percussion and auscultation, or X-ray examination.

The displacement is always toward the affected side or the side mc diseased and is caused to some extent by traction. In some instances contributing factor in the displacement is a vicariously hypertrophi lung which tends to crowd the heart toward the affected side. D placement toward the left is far more common than toward the rig side except in the far-advanced cases when the two sides are about equal involved. As a rule the displacement to the left is more pronounc than toward the right partly because of the anatomical position of t heart and partly because of the attachment of the mediastinum to t central tendon of the diaphragm. When the heart is drawn toward t left it is also apt to be pulled upward toward the axilla.

At times the right-sided displacement may be so marked as to cons tute a dextro-cardia. It is to be borne in mind that when the impu is noted on the right side in acquired dextro-cardia, it is caused by t right ventricle.

While some displacement of the heart unquestionably takes pla it is more apparent than real. This is due to the retraction of the ch wall and the consequent diminution of the capacity of the thorax whi naturally alters the landmarks with reference to the apex beat.

When the left lung is extensively diseased and retraction takes pl there is often a marked pulsation in the second and third interspaces the left. This is caused by the exposure of the right auricle which health is covered by pulmonary tissue.

Palpation serves to confirm the lack of expansion noted on inspecti and it may also serve to locate the apex beat.

The tactile fremitus will vary: at the apex over a cavity it may increased or diminished. In other portions of the chest it will be i creased or not according to the amount of underlying infiltration. rhonchal fremitus may be felt at times.

Percussion.—Assuming that there is a cavity the size of an oran in the upper part of the upper lobe with dense infiltration below it whi gradually thins out as the base of the lower lobe is reached, the followi percussion changes will be present: Over the cavity the note will be ty panitic. It is high-pitched and may have an amphoric or cracked- quality. The amphoric note is similar to that produced by percuss the cheek with the mouth open; the cracked-pot sound can be reprodu by striking the clasped and concave hands on the knee.

Wintrich's and Gerhardt's changes of note are always referred to aids in recognizing a cavity. The information furnished by these si is uncertain and of little value when present. Personally I cannot re having looked for them for years.

Below the cavity over the dense infiltration the note is dull or nea so; as the base is approached the note becomes more and more resona If the tubercles are widely separated in the inferior portion of the l the percussion note may show no abnormality.

Over the opposite lung the disease is apt to be less extensive and percussion changes correspondingly less marked. A cavity may be p ent in the upper lobe; if so the signs given above may be elicited. Ot wise, the note will be dull and gradually shade off to normal as the b is reached. A hyperresonant note may be present over the base of the least diseased lung as the result of compensating emphysema.

Percussion of the lower limits of the lung posteriorly will show the side most affected to be at a higher level.

Absolute dulness over the base of the chest in an advanced case of tuberculosis may or may not mean the presence of fluid. Not uncommonly when fluid is actually present, especially if purulent, a diagnosis of advanced tuberculosis is made. If the dulness is due to tuberculosis there will be râles and the voice sounds, if not exaggerated, will lack the distant quality so commonly present when fluid exists.

Displacement of the heart may be shown by the position of the right and left borders of cardiac dulness. If both upper lobes are extensively diseased, however, this may not be possible.

Auscultation.—The most distinctive feature of the third stage of tuberculosis is the presence of a cavity or cavities. While a cavity may or may not be present in the second it is rarely absent in the third stage although it may not be possible to elicit signs which will indicate its presence.

Inasmuch as cavitation is so universally present in advanced tuberculosis of the lungs a somewhat detailed description is necessary. It has already been shown in the section dealing with the morbid anatomy of the disease that a tuberculous cavity is usually located in the upper lobes, at, or near, the apex. In an analysis made by Ewart cavitation occurred at:

The apices	282 times.
Dorso-axillary region	227 times.
Mammary region	189 times.
Sternal region	61 times.
Base	32 times.

In 50 consecutive cases which I studied at the Phipps Institute there were in all 76 cavities present, the distribution being as follows:

Right upper lobe	15
Left upper lobe	6
Both upper lobes	30
Middle lobe (right side)	1
Left lower and both upper lobes	1

The location of the cavity with reference to the chest wall has more to do with its recognition than the size of the excavation. A small cavity, no larger than a cherry, may give signs if situated just beneath the pleura. If, on the other hand, the cavity is deeply seated it may be the size of a small orange and still give no signs. This is especially the case with cavities situated in the center of the lower lobes (Figs. 260, 261, 262).

While in most instances the approximate size of the cavity can be determined by physical signs this is not always possible. A small cavity surrounded by densely infiltrated tissue will often give the impression that there is a very large excavation. It is not possible to determine with any degree of certainty as to whether the excavation is single or consists of a number of communicating cavities. If gurgling and resonating râles are heard over a wide area a honeycombed condition of the lung is to be suspected; especially if the disease is of the acute broncho-pneumonic type in which breaking down of the lung tissue is rapid.

The character of the breath sounds heard over a cavity is i majority of instances cavernous or amphoric in quality and may be and distinct or very distant. In some instances the breath sound bronchial in quality probably owing to consolidation about the ca It is to be noted that in spite of other well-marked signs of excav the breath sounds may be almost inaudible; in very large cavities lute silence over the upper part is not unusual.

Exaggeration of both the spoken and whispered voice sounds i rule. Whispering pectoriloquy, while not pathognomonic of ca

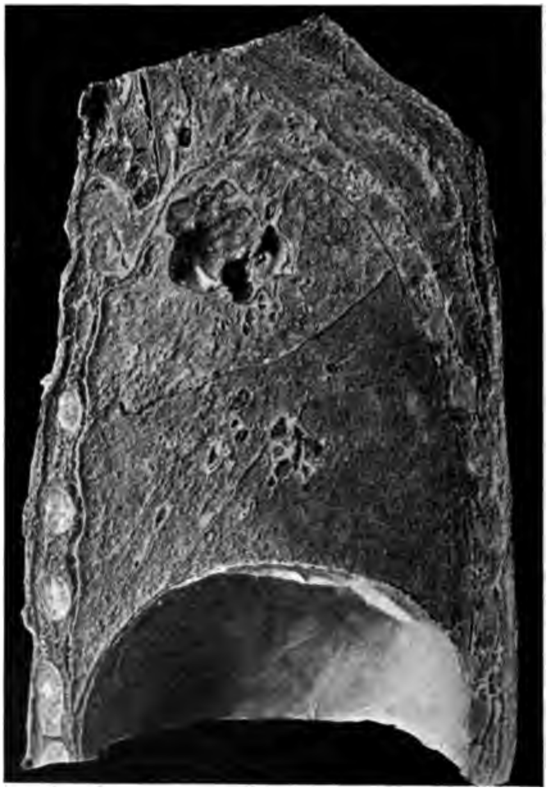
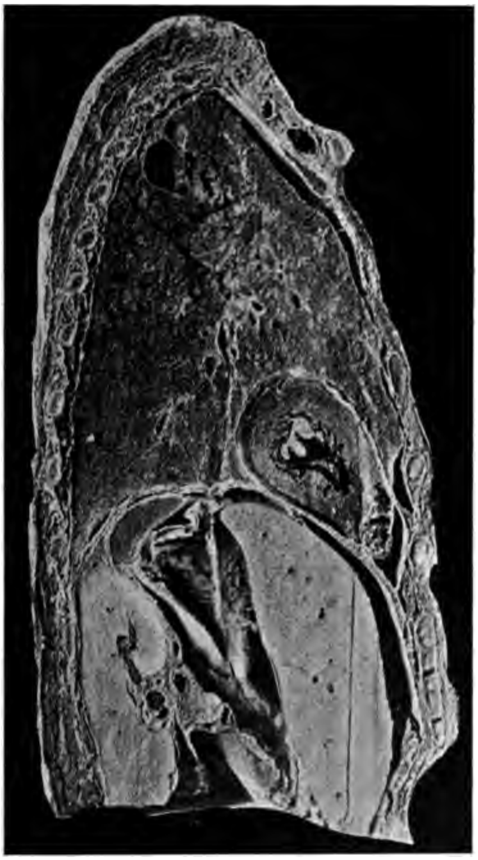


FIG. 260.—A chronic type of disease. Anteriorly just behind the clavicle is a walled-off cavity which has been formed by the coalescence of several small ones. Re der of right upper lobe and the anterior portion of lower lobe contains much fibrous

tion is the sign most constantly present. Laennec in speaking of toriloquy (and the same applies to whispering pectoriloquy) states the intensity and perfection of this phenomenon ranges from that w is unmistakable to that which is of doubtful import. "The cir stances that concur to render pectoriloquy perfect are: complete e ness of the excavation, increased density of the pulmonary tissue for its walls, its easy communication with one or more bronchial tub some magnitude, and its proximity to the parietes of the chest."

he sound produced by coughing very often has a metallic or amphoric ty and when present is very suggestive of excavation.

âles may or may not be heard over the excavation. In chronic scent cases the cavity may be dry and no râles will be heard. In ve cases râles are almost always heard and are often metallic or nating in quality. In my experience the presence of *râles having a llic quality is the most certain sign we possess.*



1.—Cavity in right upper lobe posteriorly. Physical signs elicited posteriorly Rest of right upper lobe air-bearing but contains a good deal of fibrous tissue.

a clinical and post-mortem study of cavities I found that in 50 here were present 76, of which 58 were recognized during life. The ping detection were, for the most part, small and centrally located. s the value of the various signs on the frequency with which each ed, the following results were obtained:

		Per. Cent
Whispering pectoriloquy	53 out of 58	(91.3)
Tympany (on percussion)	39 out of 58	(67.4)
Cavernous or amphoric breathing	38 out of 58	(65.5)
Gurgling or consonating râles	33 out of 58	(58.6)

Although none of the above signs are pathognomic of a cavity presence of any one of them is suggestive and the probability of its ex ence is increased by combinations of any two or more of them. T sources of error are to be borne in mind: (1) Consolidated pulmon tissue about a bronchus may give rise to the same phenomena. T mistake was made in five instances in the series I studied. (2) The si of a single cavity at the right or left apex may be transmitted across spine and lead to a diagnosis of a cavity at both apices. This er can be avoided by tracing the sounds from their origin when they v be found to diminish in intensity the farther the bell of the stethosc is moved from the site of the cavity.

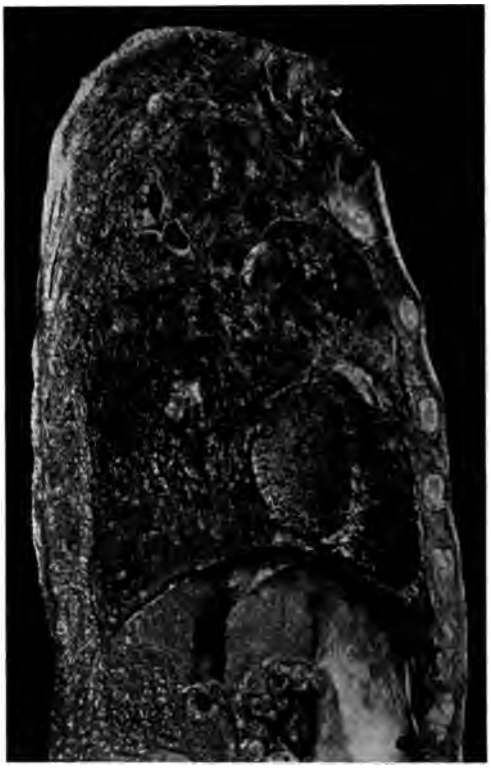


FIG. 262.—Two small cavities situated at some distance from the posterior pleura. Dee seated cavities are apt to escape detection by physical signs.

The auscultatory signs over the remainder of the chest para those of percussion. Below the cavity the infiltration is apt to be de and the breath sounds bronchial or broncho-vesicular in character. latter type of breathing is usually present over the base of the side n affected and, in addition, may be more or less suppressed. The v sounds are more or less exaggerated depending on the density of infiltration.

Over the opposite lung there may be signs of a cavity at the apex evidences of infiltration beneath it but rarely as well marked as on side most diseased. Over the lower lobe the breath sounds are usually puerile in type due to compensatory emphysema. Although at the autopsy table scattered tubercles are usually found in the emphysematous lower lobe they give no evidence of their presence during life. They probably represent, for the most part, a terminal infection such as occurs in the other viscera.

In the advanced stage tachycardia is the rule, and the muscle tone of the first sound is poor. Accentuation and reduplication of the second pulmonic sound is common; less frequently these changes are noted in the first sound. Functional murmurs are frequent and are generally heard at the pulmonic area. Irregularity of the cardiac rhythm is noted in a few instances.

Diagnosis.—The recognition of pulmonary tuberculosis in its different stages must take into account four factors:

1. A clear conception of the morbid anatomy of the disease; the location of the primary lesion; how it first manifests itself; the "line of march" as it progresses; and finally the character of the lesions it produces.

2. There must be a proper appreciation of the significance of the symptoms and sufficient moral courage to act promptly even when the appearance of general good health seems to deny the presence of serious trouble. It should never be forgotten that the onset is usually insidious and the early manifestations indefinite. To delay the diagnosis until the evidence is unequivocal is, only too often, to let slip the opportunity most favorable to restore health.

3. It cannot be too strongly emphasized that the diagnosis of pulmonary tuberculosis, particularly in the first stage, is not to be based exclusively on the presence or absence of physical signs. "Invaluable as they are, their importance is greatly enhanced by association with symptoms and the knowledge of pathological laws. Not only are we to avoid exclusive reliance on the presence or absence of physical signs, but it is also to be borne in mind that physical signs, while often indicating the extent and degree of pulmonary damage, convey no direct information as to the pathological nature of those changes."

4. It is necessary to employ in every case one or all of the available laboratory aids. There can be no excuse for neglecting the examination of the sputum. In many instances this may seem superfluous. It is to be borne in mind, however, that there are many conditions which simulate both the symptoms and physical signs of tuberculosis and are only to be distinguished from it by examination of the sputum.

The significance of the tuberculin tests will be discussed later. The advantages and disadvantages of the Roentgen rays are also considered elsewhere. A review of the literature on the subject of complement fixation in tuberculosis shows that while the test is positive in a large proportion of cases it is, at present, not of much practical service.

[redacted]**is of Healed and Arrested Lesions.**—Very frequently one
ndividuals with abnormal signs at one or the other apex. In
the question will arise: Is this an incipient lesion or is it the
uble which has occurred in the past and has undergone
cure?

ical signs are not conclusive. The clavicle may be unduly
nd slight flattening with restriction of expansion may be
h the clavicle, indicating some shrinkage at the apex. The

breath sounds may be slightly suppressed or broncho-vesicular in type. The voice sounds are, as a rule, exaggerated. There are no râles.

These signs differ in no respect from those found in the early, active stage of the disease. In such cases the question as to whether the lesion is active, or healed and quiescent, will rest on the presence or absence of symptoms. If there is no cough or expectoration, no fever, no loss of weight and there has been no evidence of blood in the sputum or an attack of pleurisy within the past year or two, one may assume, with reasonable certainty, that the signs are indicative of an old tuberculous infection which during its active stage was so mild as to escape notice. Occasionally signs, such as those mentioned above, may be caused by non-tuberculous infections.

The subcutaneous injection of tuberculin has been advised to distinguish between a healed and an early active lesion. If healed there may be slight constitutional reaction (fever, malaise, joint pains) but no focal reaction; the latter consists of râles over the suspected area and the development of a slight cough with possibly some expectoration in addition to the constitutional symptoms. The use of tuberculin as a diagnostic agent will be considered in another place.

Another type of case is that in which there are evidences of extensive pulmonary damage and in which the patient has some cough and expectoration but no constitutional symptoms. Such a case may or may not have been under treatment when first seen. If one judges by the physical signs alone the patient may be considered to be in a serious condition and may be advised to abandon his business and enter a sanatorium at once. The course to be pursued will depend entirely on the general condition of the patient's health and the presence or absence of symptoms denoting activity of the disease.

A woman whom I have had under observation for ten years has at the present time the same physical signs as when originally seen, namely, signs of a cavity at each apex and impairment of the percussion note, feeble broncho-vesicular breathing and fine râles extending down to the fourth rib on the right side. Her general health is excellent, although she has an occasional hemoptysis, and she lives the life of a normal individual. Twice within the past two years, because of an attack of blood spitting, she has consulted men of large experience in tuberculosis work. In both instances she was given an unfavorable prognosis and advised to enter a sanatorium at once. Cases such as this one are not infrequently seen and illustrate very clearly that entire dependence cannot be placed upon the physical findings alone. One of the peculiar features of tuberculosis is that very often with the existence of extensive pulmonary damage the symptoms are negligible and the patient enjoys fairly good health; on the other hand, physical signs may be very slight or even absent, and the symptoms will be of the most serious nature.

Examination of the Sputum.—The examination of the sputum for tubercle bacilli is a very simple procedure, the details of which are familiar to every one. While the presence of tubercle bacilli in the sputum is absolute proof that a pulmonary tuberculosis exists, their absence is no assurance that the patient is free from the disease. *In the earliest stage of tuberculosis the sputum will be found negative for tubercle bacilli in from 60 to 75 per cent. of cases* It is most important that this fact should be known. Many practitioners fail to realise this and are satisfying both themselves and the patient that tuberculosis is not present as the result of one or two negative examinations. I repeat, therefore, that in the early stage of the disease, one, or even half a dozen, negative examinations mean nothing.

Another very common error is that of assuming the case to be one of advanced pulmonary tuberculosis because the symptoms and physical signs seem to clearly indicate it as such. Inasmuch as the diagnosis seems certain the sputum is not examined. It is because of this neglect that many cases of bronchiectasis, pneumoconiosis, chronic empyema, the mycotic infections and even chronic cardio-renal disease, are mistaken for tuberculosis. A single negative examination in a case with extensive pulmonary damage demands that the examination be repeated until tubercle bacilli are actually found or until a sufficient number of negative examinations make it clear that some process other than tuberculosis is present. While in the majority of instances the presence or absence of tubercle bacilli is all that need be determined, one should always be alive to the possibility of other sources of infection.

The conditions which simulate tuberculosis and which are recognizable by the examination of the sputum, although not numerous, should always be kept in mind if tubercle bacilli cannot be found. In default of any demonstrable organism, such as the tubercle bacillus, actinomyces, blastomyces, aspergillus, ova of the lung fluke, etc., the pulmonary trouble may be due to malignant disease, syphilis, or some chronic inflammatory condition such as cirrhosis of the lung or pneumoconosis. If one is not familiar with the bacteriological characteristics of the rarer forms of infection the sputum should be sent to a competent bacteriologist for examination.

Of the various infecting agents which produce lesions similar to tuberculosis there is only one that bears any resemblance to the tubercle bacillus, namely, certain forms of the streptothrix group. To those who are not thoroughly familiar with the morphology of the tubercle bacillus small acid-fast rods may be mistaken for Koch's bacillus. The *streptothrix* may occur as an independent organism but is more often encountered as part of the mixed infection in cases of tuberculosis with cavity formation and in cases of bronchiectasis. It is to be borne in mind that the bacillus-like rods of the streptothrix may occur in chains instead of being clumped and that they are decolorized with 30 per cent. nitric acid. They are often resistant to 20 per cent. sulphuric acid and Gabbet's stain.

At one time it was believed that the number of tubercle bacilli present in the entire slide or if numerous, the average number per field, was of value in determining the probable outcome of the disease. It is now recognized that the number of bacilli per field at the time of the first examination is no indication of either the severity or the extent of the disease. From 40 to 50 bacilli to each field may be present in an mild case; on the other hand, the bacilli may be very few in in a case which begins with severe symptoms and progresses to a fatal termination. In a general way a gradual diminution umber of bacilli at each successive examination, is of favorable And on the other hand, a rapid increase in the number is apt indication that the process is extending and the lung tissue is down. Much more reliable indications as to how the disease is behaving are to be found in the patient's general condition, the abatement of symptoms and stationary or retrogressive physical signs.

The examination of the sputum for tubercle bacilli is so simple a procedure that the statement that they have been found in the sputum is usually accepted as being true. In common with other methods of diagnosis, however, the reliability of a sputum examination depends to some extent on the experience of the examiner. Unless my own experience is unique, I am inclined to believe that not a few positive reports are being made which in reality are negative. Within the past few years I have seen five cases in which tubercle bacilli were said to be present in the sputum. In one case the autopsy failed to show any evidence of tuberculosis. In the others a most thorough study of the sputum, including animal inoculations, failed to show the organism. In addition the subsequent history of the patients made it evident that a mistake had been made. If there is any doubt as to the ability of the examiner one should examine the sputum oneself or send it to a reliable laboratory.

In patients in whom tuberculosis is suspected but who have no sputum, the administration of *iodide of potassium* has been recommended. This procedure cannot be condemned too strongly. As it is now quite generally recognized that iodide of potassium is very apt to cause a breaking down of tuberculous foci in the lungs there is little to be said in favor of using the drug for diagnostic purposes. By so doing a quiescent lesion which is giving no trouble may be aroused into activity.

Tuberculin Tests.—The accepted belief at the present time is that a positive reaction to one of the tuberculin tests is indicative of a tuberculous lesion somewhere within the body. A positive test does not mean, however, that the individual has clinical tuberculosis. It must be clearly kept in mind that there is a *great difference between tuberculosis that is clinically recognizable and hypersensitiveness to tuberculin.* Hypersensitiveness is extremely common, and is encountered in a large proportion of healthy people, the frequency with which it is met increasing rapidly from the second year of life. It is a great mistake to assume that because an individual reacts to some one of the tuberculin tests that he is tuberculous in the sense that he needs active treatment; and, as I shall later point out, a negative tuberculin test does not entirely free us from responsibility.

In regard to the tests themselves there are certain facts which should be kept in mind. First, as regards the *subcutaneous test.* At one time the test was extensively used in adults and a positive reaction was, and by many still is, regarded as conclusive proof that the individual has tuberculosis. Of recent years, however, some observers have warned against its use, pointing out that the test is capable, in certain instances, of stirring an inactive lesion into an active one. Furthermore, there is now a tendency not only to limit the use of the test, but in addition to ignore the constitutional symptoms of a reaction (such as fever, joint pains, local reaction, etc.) and to call positive only those cases which give a focal reaction, such as râles at a suspected apex, or some pain, redness, and swelling in a suspected joint.

The *cutaneous or von Pirquet test* has achieved enormous popularity by reason of its simplicity, and also because it is widely credited with being a positive indication of tuberculosis. In children under two years of age it furnishes valuable evidence when positive and usually means linical tuberculosis. Beyond the second year, however, it loses much f its positive value, and the older the child the more unreliable it is s an evidence of true tuberculosis.

The *percutaneous* or so-called Moro test has about the same gnificance.

Finally, we have to consider the *conjunctival test* introduced by olff-Eisner and Calmette. The majority of observers have come to gard this test as dangerous, because of the possibility of producing an flammatory condition which may lead to serious damage of the eye. ccording to Hamman and Wolman,[1] who have employed the test in a rge series of cases, the test is free from danger, and is superior to the oro and von Pirquet tests in that it is not so sensitive and that a posi-ve reaction points strongly to an active rather than a latent process, pecially so when supported by other indications.

The question naturally arises: What is the practical value of the von irquet or Moro test in determining the individuals who have true berculosis and those who can be disregarded? We are forced to con-ude that they are of little value except as an evidence of hypersensi-veness. In a series of children studied by Kaufman and myself[2] we peatedly saw anemic, under-nourished children with or without tuber-lous parents who gave no reaction; and, on the other hand, we en-untered children who presented no physical signs; who were in robust ealth, and who had violent skin reactions.

The X-ray in the Diagnosis of Pulmonary Tuberculosis.—The ques-on of the relative merits of the X-rays and the ordinary method of hysical examination in incipient or early tuberculosis is still *sub judice.* linicians are not in agreement as to the superiority of the newer over the lder method and between the clinicians and the roentgenologists there a wide breach. The latter, with few exceptions, arrogate unto them-elves an ability to recognize early tuberculous lesions in the lungs which not warranted. In the first place the earliest manifestations of tuber-ulosis as shown on the roentgenogram, are not distinctive. Many imple chronic infections will give the same picture. Again, as Friedrich üller, Holzknecht and others have pointed out a deposit of a few early ubercles with accompanying catarrh, and which constitute an anatom-ally incipient case, will not cast a shadow on the plate. Active dry leurisy also gives no hint of its presence whatsoever, so far as the oentgenogram is concerned.

Many roentgenologists claim that an early tuberculous process is istinctive without realizing that other infectious processes of the lung, f whatever character, generally spread along the course of the air assages. As Baetjer[3] has pointed out, these become inflamed and, s in all inflammatory conditions, they produce reaction, consequently he fibrous sheath which surrounds the bronchi, artery, vein and lymph hannels becomes thickened. It is just this type of cases that it is so mportant to make an etiological diagnosis. Although the clinical evi-ence and the physical signs in such cases may be inconclusive, the entgenogram is equally so, for while changes may be shown in the plate is impossible to state whether they are due to tuberculosis or some other infection. It is only when the tuberculous lesion has become sufficiently advanced that a definite opinion can be given as to its nature. When this point has been reached an appeal to the roentgenogram is not necessary as all the signs and symptoms of the disease are apparent from the clinical side.

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[1] "Tuberculin in Diagnosis and Treatment," 1912.

[2] *Am. Jour. of Med. Sc.*, October, 1914.

[3] *International Clinics*, Twenty-sixth Series, vol. iii, 1916.

Another disputed point is the question as to whether it is possible by roentgenography to determine whether a tuberculous lesion is active or inactive. Many roentgenologists claim that such a distinction can be made. Baetjer takes the stand that such a differentiation cannot be made, and in this opinion I concur. The activity or inactivity of a tuberculous lesion is to be determined by symptoms and neither physical signs nor radiography can determine this point.

At the present time the use of the X-ray is too recent to determine what its ultimate status will be. It is fair to assume, however, that those who now oppose it will eventually take a more liberal view and, on the other hand, it is equally certain that roentgenologists will have to modify their claims. Already there are signs pointing to the latter view. Not so long ago tremendous importance was attached to root shadows. Now we hear less about these "visionary interpretations," as Osler termed them. The elimination of root shadows has been brought about largely by improved technique, chiefly in the substitution of the stereoscopic for the single plate.

What is needed beyond everything else to establish the true status of the X-rays in pulmonary disease is the anatomical corroboration of such findings. Anatomical proof was the foundation upon which the art of physical diagnosis was reared. Until this proof is forthcoming it is inevitable that differences of opinion will occur. Such proof can be furnished only by combining the observations of the clinician, the radiographer and the pathologist. An investigation of this kind "requires the most patient following up of cases, it may be over long periods, in the hope that even one single case, which has been carefully and accurately investigated clinically and radiographically, will reveal itself to the pathologist" (Barty King).

Although the value of the X-ray in the diagnosis of early tuberculosi still remains to be proved, there can be no question as to its advantage in showing the extent of the lesion and in the detection of encysted empyema or a partial pneumothorax. Minor[1] after years of experience has found the fluoroscope of service for this purpose. By its use th topographical distribution of the lesions can be made out readily and i addition collections of pus or other obscure conditions can be detected This method has the advantage of not requiring special training as doe the making of the stereoscopic plates.

CHRONIC TUBERCULOSIS IN EARLY LIFE

Morbid Anatomy.—It must be borne in mind that the clinical forms o human tuberculosis in different ages possess correspondingly differen anatomical features, a knowledge of which is necessary for the understanding of the interpretation of physical signs. In the majority of instances, tuberculosis, when it involves the lungs of infants and youn children, is bilateral, widespread (either of the miliary or broncho-pneumonic form), and acute in character (see "Reinfection," p. 306).

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[1] *International Clinics*, Twenty-sixth Series, vol. ii, 1916.

In the adult, on the other hand, the disease begins at one or the other apex, follows a fairly definite course in its progress, and is chronic in character, although subject to periods of acute activity. As a rule the clinical manifestations of tuberculosis as they occur in infants and young children differ from the adult type, and this is not altered by the fact that the juvenile type may occur in adults, and *vice versa*.

Symptoms.—The symptomatology of tuberculosis in adults, while subject to many vagaries, is invaluable in making a diagnosis. Indeed in many cases of incipient tuberculosis it is our chief reliance in determining the nature of the trouble. In children, on the other hand, we are deprived of much of the value of symptoms, because of the inability of young children to give a correct description of their trouble; nor, as a rule, can the parents throw much light on the question.

One of the most reliable means of detecting early tuberculosis in the adult is the slight rise in temperature which may occur daily or only every few days. In children, however, little reliance can be placed in these slight elevations of temperature, for the reason that growing children normally have a higher temperature than adults. Landois[1] gives as the normal limits 99.5° to 100.1°F. (37.87° to 37.62°C.) in children from five to nine years. The reason for this is, in all probability, a more active metabolism, although other influences of undetermined origin may also, in part, be contributory.

Physical Signs.—In taking up the question of physical signs in the chest of infants and young children we have to consider what is probably the most difficult phase in the art of physical diagnosis. And while it is true that the underlying principles which enable us to detect disease in the chest are the same, whether it be adult or child, there are certain differences between the two which must be kept in mind. Generally speaking the man whose training has been confined to the examination of adults alone, or children alone, is likely to draw false conclusions when he invades one or the other field, and much that has been written regarding the presence of chronic tuberculous lesions in the lungs of children has been contributed by those whose standards have been obtained by the examination of adults with tuberculosis.

It is not altogether easy to fix definitely the age period at which the signs peculiar to children cease. Some have arbitrarily fixed this limit at the age of six years, although from my own experience ten years would be more nearly correct.

Probably the most distinctive difference between the adult and the child is that in the latter all of the sounds are exaggerated. Not only is the breathing of the familiar puerile type, but all the vocal sounds are increased, and in addition the percussion note is commonly hyperresonant.

As an illustration of how the exaggeration of physiologically normal signs may be misinterpreted in the child it is interesting to note that 79 children out of 362 studied at the Phipps Institute, who were charted
ormal physical signs no less than 67 had impairment and
lar breathing at the right apex. It will be recalled that
he right apex normally has a slightly impaired note, pro-
on, and increased fremitus, and that the explanation of
l out by Fetterolf[2] as being due to close proximity of the

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t-book of Human Physiology," 10th ed., p. 391.

Int. Med., Feb., 1909.

trachea to the right upper lobe. The very high proportion of cases in this group, with apparent disease at the right apex, would seem to indicate that what was in reality a normal finding was interpreted as being pathological.

One's belief in this is strengthened from the result of reports of 111 of these children three or four years after the first observation. Of these 111 cases 17 were noted as having a diseased right apex, and in 2 more both apices were supposed to be affected. At the time they were investigated 17 of the 19 were noted as being in good health, 1 had died of tuberculosis, and 1 still had some cough. By some, the presence of abnormal physical signs at the right apex is believed to be due to enlargement of the tracheo-bronchial lymph nodes.

In addition to the exaggeration of the breath sounds already noted the respiratory sounds in children are subject to a number of vagaries, and the younger the child the more difficult is the interpretation of the auscultatory signs. The expired air may, for instance, be directed into the pharyngeal vault or against the roof of the mouth, with the result that a bronchial quality of breathing is heard all over the chest. This can be obviated if the child is old enough to learn how to breathe properly. Then again in young children and infants the lungs at times seem to move independently of one another, with the result that loud puerile breathing is heard over one side, while over the opposite side the breath sounds are almost inaudible, and this condition shifts from side to side. This peculiarity is often due to faulty posture. If care is not taken to see that the child sits straight, and that one or the other side is not held tense, not only will there be very faint or nearly absent breath sounds on the cramped side, but in addition the percussion note may be impaired, or indeed absolutely dull. I have often demonstrated this fact to students and shown them how the signs on the two sides can be almost instantly reversed. This fact is worth remembering, as the unwary may make a diagnosis of pneumonia or pleural effusion.

Lymph Nodes.—In nearly every article dealing with the diagnosis of tuberculosis in children great stress has been laid on the presence of palpably enlarged cervical lymph nodes. Indeed in not a few instances, in which surveys of large groups of children have been made, this apparently has been the only evidence on which the diagnosis was made. The fact has been ignored that the age of childhood is likewise known as the lymphoid age, and that all children, irrespective of their social condition, have lymph nodes which are readily palpable. The enlargement of the lymph nodes is not readily detected until about the second year, but from then on until shortly before or after the age of puberty this is the case, although as the child approaches adolescence certain groups, such as the epitrochlear, the axillary, and the inguinal, tend to shrink in size and become less easily detected. The submaxillary node at the angle of the jaw is usually the largest under normal conditions, and is usually the last to shrink to a size that it can no longer be palpated.

To confine the examination to one group, such as the cervical, and because the nodes in this situation are palpable, to assume that it is an evidence of tuberculous infection does not seem in any way justifiable, especially so in view of the fact that all the lymph nodes of the body are hypertrophied according to adult standards.

If there exist in the neck a single large node or a tumor mass made up of a number of nodes, which are matted together and adherent to the rrounding tissues, the presumption is strong that we have to deal with uberculous adenitis, and especially so if there is evidence of softening, the overlying skin is inflamed. This, however, is true scrofula, and s nothing to do with nodes which vary in size from that of a bean to split pea, or smaller, which are freely movable, and which manifest ither softening nor inflammation.

Ten years ago children who were definitely classed as being tuberlous are to-day considered as belonging to the so-called *pretuberculous* oup. Such children are usually anemic, under-nourished and undered; the lymph nodes are easily palpable and the von Pirquet or Moro in tests are, as a rule, positive. The type is a familiar one among the ildren of the poorer classes although by no means limited to them. the great majority of these children the evidences of ill health dispear if the living conditions can be improved and above all if an adeate dietary can be obtained. The open air school has demonstrated ry clearly how quickly these children react to improved sanitary nditions.

TUBERCULOSIS OF THE TRACHEO-BRONCHIAL LYMPH NODES

Of recent years much has been written on the subject of enlargement the mediastinal lymph nodes. As I have already stated a diagnosis chronic tuberculosis of the lungs in children is now far less common than s formerly the case. At the present time children presenting obscure mptoms which may be tuberculous in origin, are considered by many be suffering from a localized infection of the tracheo-bronchial lymph des. The majority of observers consider that the initial lesion in ildren is situated in these nodes and that the invasion of the lungs is condary. Ghon,[1] on the other hand, places the initial lesion in the lung. is his belief that the bronchial lymph nodes are secondarily invaded. nong young children and infants who succumb to tuberculosis, infecn of the mediastinal lymph nodes is a common finding. The disease y become so extensive as to cause death without spreading to other uctures. More frequently, however, the disease invades the lungs or comes widely disseminated. If the child survives this initial infecn, the process in the majority of instances becomes arrested. In ildren over three years of age fibrous and calcareous changes very quently take place.

Symptoms.—The symptomatology of this condition is very indefinite. the majority of cases in which there is reason to believe that the cheo-bronchial nodes are tuberculous the child is anemic, frail and viously out of health. Susceptibility to acute colds may in some tances be due to enlargement of these nodes. There may be a brassy gh, some dyspnea and at times thoracic pain, especially in the interpular region. Rupture of an enlarged gland into one of the surrounding uctures occasionally occurs.

Physical Signs.—The physical signs are quite as inconclusive as the nptoms. Enlargement of the veins over the upper part of the chest cited as one of the evidences of enlarged mediastinal glands. On cussion dulness in the first and second interspaces close to the stern and dulness over and to either side of the upper thoracic vertebra is ascribed by most authorities as being quite constantly present. T percussion changes are usually more marked on the right side. (auscultation Eustace Smith's sign is occasionally encountered but tl frequency of venous hums at the root of the neck in healthy childr renders this sign of questionable value.

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[1] "The Primary Lung Focus of Tuberculosis in Children," Trans. by Barty King,

The one sign upon which the most emphasis has been laid is th described by D'Espine.[1] This sign consists of a prolongation of t whispered voice over the upper dorsal vertebræ; occasionally it is hea as low as the eighth dorsal vertebra. It is elicited by having the patie whisper "one, two, three," "three, thirty-three" or repeat the wo "tree." The distinctive quality of the sign is the prolongation of t final "e," for an appreciable time after the voice stops. Some belie D'Espine's sign is pathognomonic of enlarged tracheo-bronchial lym nodes; others, while attaching great importance to its presence, st that it is heard at times, in the absence of any such enlargement. Mor in a study of 666 children of the wealthy and well-to-do classes was a to elicit the sign in but 40 (6 per cent.).

Although not infallible, an X-ray examination is the most cert method of detecting enlargement of this group of lymph nodes.

Diagnosis.—Occasionally a child is seen in whom the symptoms, physical signs and the radiograph point almost conclusively to this c dition. But in most instances neither the symptoms nor the physi signs are distinctive. In delicate children, and especially in those w give a history of exposure to the disease, an X-ray examination should made. It is to be borne in mind, however, that any of the acute inf tions are apt to cause enlargement of the lymph nodes and that t enlargement may persist. It is, therefore, evident that while the X-r will show enlargement of the lymph nodes they do not indicate etiological factor.

Although the diagnosis of enlargement of the mediastinal lym nodes in children is a common one, my own experience, checked up radiographic examinations, is that the condition is less frequent th most observers claim and that the enlargement, when present, lacks tl serious significance many authors attach to it.

ACUTE TUBERCULOSIS OF THE LUNGS

Galloping Consumption, Phthisis Florida.—In contrast to the chron type of the disease pulmonary tuberculosis occurs also in an acute for of which there are two types, the penumonic and the broncho-pneumoni In the chronic form of the disease the progress is slow and the duration the disease usually is measured by years. In the acute types, on the oth hand, the invasion is rapid and widespread and may end fatally with a few weeks, particularly when it occurs in children. In adults the cour of the disease is more often subacute and may be prolonged for five or s months or even longer. Occasionally what promises in the beginning be an acute process subsides and undergoes arrest or develops into tl chronic ulcerative type of the disease.

The Broncho-pneumonic Form

This is the most frequent and the most characteristic form of tub culosis in infants and young children, and is the one which at this a usually causes death (Holt). Acute tuberculosis is not uncommonly the sequel to an attack of measles or whooping cough. In negroes, acute or subacute pulmonary tuberculosis, particularly of the broncho-pneumonic type, is relatively common. Among adults of the white races acute broncho-pneumonic phthisis is relatively infrequent. It is most apt to occur in young females or in those with a strong hereditary predisposition.

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[1] *Bull. Acad. de Med.*, 1907, lviii, 142–146.

[2] *Am. Jour. Dis. Children*, April, 1916.

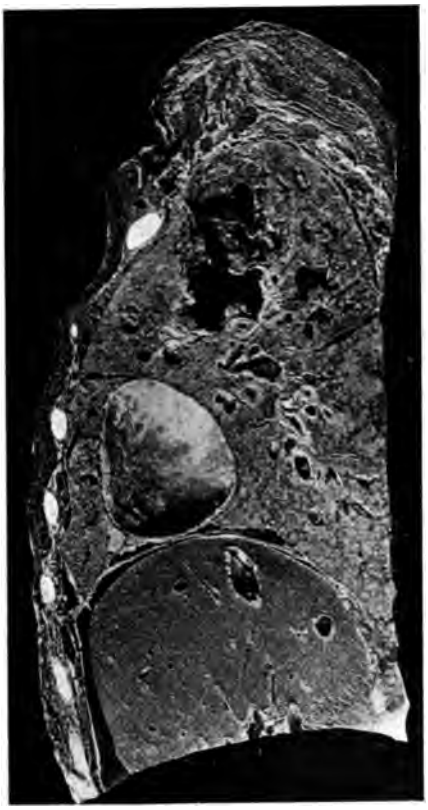

FIG. 263.—This specimen obtained from a young colored girl shows the acute broncho-pneumonic type of disease. Lung breaking down rapidly and forming large ragged cavities. Dense infiltration in lower lobe.

In cases of chronic ulcerative tuberculosis an acute broncho-pneumonic process is not infrequently engrafted upon the chronic process as the result of the aspiration of the contents of a cavity or the aspiration of blood following an hemoptysis.

Morbid Anatomy.—The commonly accepted teaching has been that in children the primary focus is one of the peribronchial lymph nodes and that infection of the lung is brought about by the rupture of a caseous area into a bronchus near the hilus. Ghon, however, asserts that the reverse is true. He believes, as a result of careful anatomical studies, that the primary focus is in the lung and that the peribronchical nodes are involved secondarily. In children both lungs are involved but usually one is muc more so than the other.

In adults an old quiescent focus or partially healed cavity may suddenly become active and rapidly invade a portion of or all of one lung On section the lung is moist and seen to be studded with areas of variou sizes which are usually ranged around the bronchi "like clusters of grape about the stem." The patches may be very small in which case they ar gray or semitranslucent, or they may be quite large as the result of th coalescence of several areas. The latter are usually grayish red o yellowish white in color.

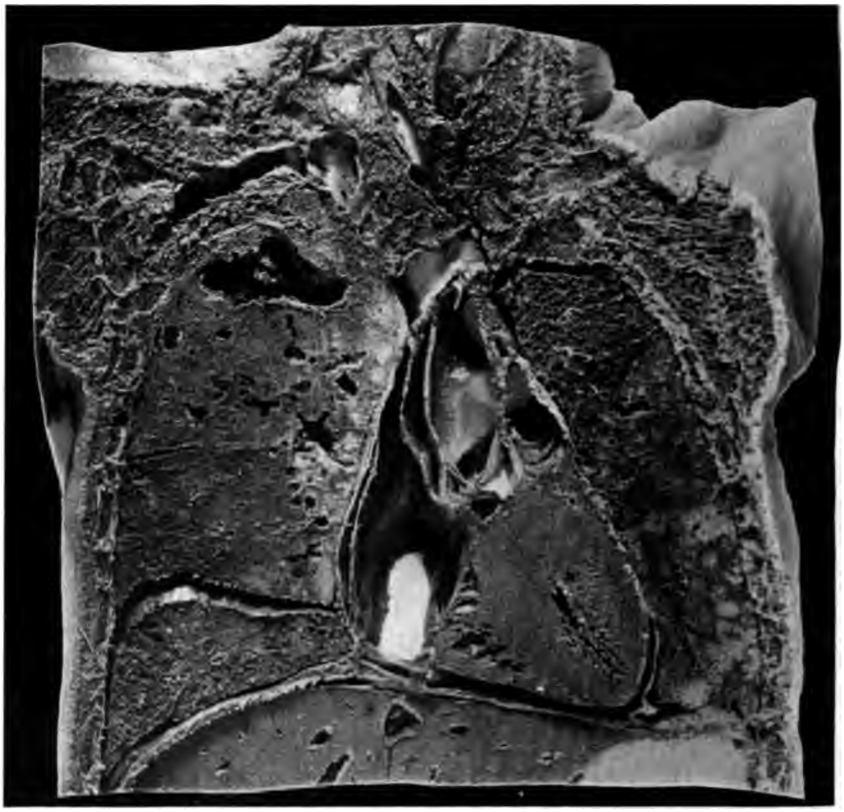

Fig. 264.—Broncho-pneumonic type of tuberculosis. **Rapid breaking down of tissue in right lung. Chronic fibroid condition at left apex which is probable source of infection.**

The fusion of a number of contiguous patches often gives rise to larg caseous foci in which softening takes place very rapidly. In the ver acute cases death may ensue before softening and breaking down take place but in the subacute cases cavity formation is usually the rule Between the caseous areas the lung tissue may be crepitant or it may b grayish red and infiltrated with a serofibrinous or gelatinous materia (gelatinous pneumonia).

The disease spreads rapidly. The individual foci increase in size and by coalescing with contiguous patches form large caseous areas which show little or no tendency to fibrosis. As a result caseation, softening and the formation of ragged, irregular-shaped cavities are common (Figs. 263 and 264).

Symptoms.—The disease may manifest itself abruptly with a chill or a succession of chills. The temperature rises quickly and the patient has every appearance of being acutely ill. In children the initial stage differs hardly at all from that seen in ordinary broncho-pneumonia. At times the first evidence of trouble is an hemoptysis. More often the onset is more or less insidious. The patient has a slight cough, some elevation of temperature, malaise and pains in the limbs. In addition he has no appetite, feels weak, and has no ambition to go about his daily duties.

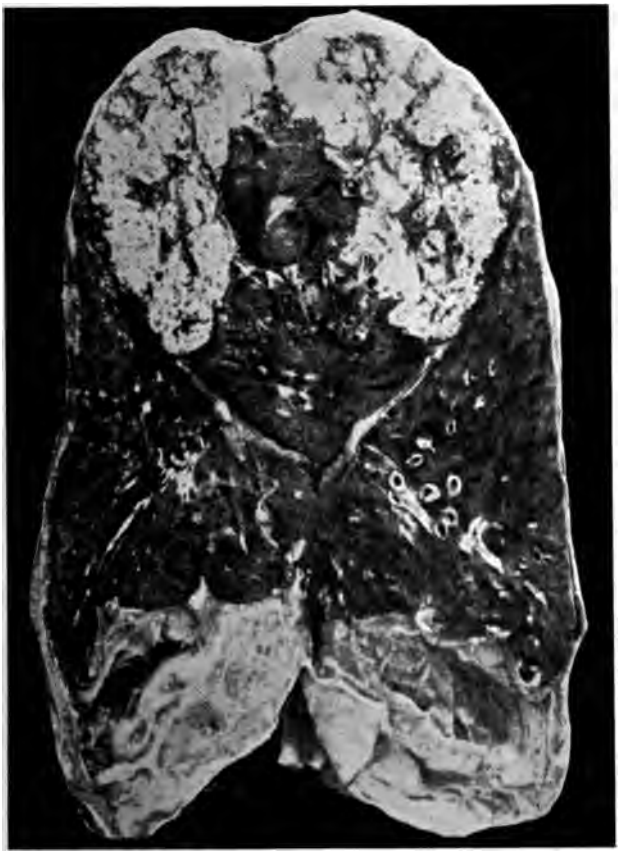

Fig. 265.—Caseous pneumonia.

In a relatively short time the symptoms become well marked. The temperature becomes high and continuous in type with an occasional intermission or remission (see Figs. 247 and 248), the cheeks have a hectic flush and the skin becomes burning hot. Emaciation is usually very marked and within a few weeks the patient may be but a shadow of his former self. Severe toxemia manifests itself by chills and drenching sweats. The cough becomes progressively worse and the sputum more and more abundant. The latter is at first mucoid but soon becomes yellowish or greenish in color and may be streaked with blood. Tubercle bacilli are usually abundant.

As the disease progresses the pulse becomes more and more rapid and the respiration is also greatly accelerated.

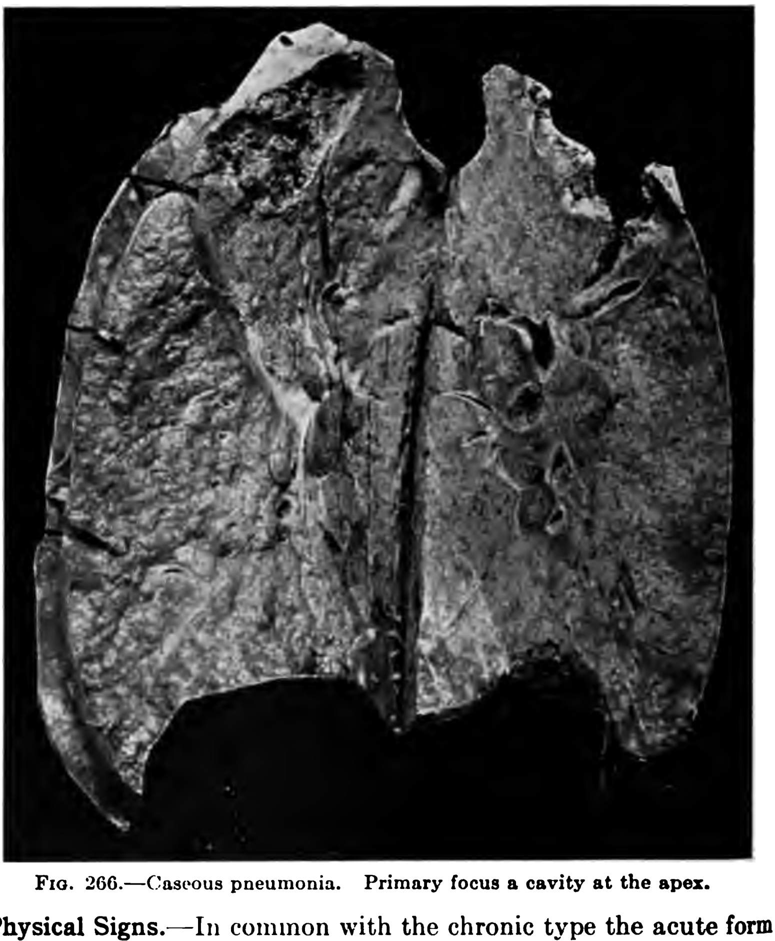

Fig. 266.—Caseous pneumonia. Primary focus a cavity at the apex.

Physical Signs.—In common with the chronic type the acute form of the disease almost invariably appears first at the apices or if a large portion of the lung is invaded the signs are most marked at the summit of the lungs. At the beginning râles such as occur in acute bronchitis may be heard over one or both lungs. Sooner or later the evidences of infiltration become apparent. There is dulness on percussion, increased tactile fremitus and broncho-vesicular breathing. Mixed râles are heard especially the fine crackling variety after coughing. Quite often there so present a pleural friction rub. If the caseous areas break down empty through a bronchus the usual signs of cavity are present.

n children presenting the symptoms of ordinary broncho-pneumonia well to remember that a preponderance of the physical signs at apices or high in the axillæ point to a tuberculous origin of the ble.

The Pneumonic or Lobar Form

Acute pneumonic phthisis is a rare condition. The subacute form, ever, is relatively common. The latter may terminate fatally in the se of a few months; the acute symptoms may subside and the lesion ain localized; or it may pass into the chronic progressive type.

The lobar type of the disease is very rare in children being met with, rule, in adults. While it may have its origin in a tuberculous focus ide of the lungs the usual starting point is an old chronic lesion at apex which suddenly becomes active.

Morbid Anatomy.—The lesion may involve a portion of the lobe . 265), an entire lobe or even the entire lung. The affected portion be completely consolidated there being no healthy pulmonary tissue aining. The weight of the lung is greatly increased. Section through diseased lung shows large caseous areas which are smooth, dry, air- and of a yellow, grayish-white or yellowish-white color. The affected ion cuts like a piece of cheese. In some instances the cut section is ular and presents a grayish-white mottled appearance due to con- erate caseous areas and consolidated hyperemic patches of pul- ary tissue. Just as in the broncho-pneumonic form there is little o evidence of the formation of fibrous tissue. This is especially ceable if a cavity forms. Instead of a limiting fibrous membrane, as is seen in the chronic type of the disease, the wall is composed of ed, necrotic tissue (Fig. 266).

The overlying pleura usually shows inflammatory changes and a nous exudate. The peri-bronchial lymph nodes are swollen and n caseous.

Symptoms.—The onset in this type of the disease is nearly always pt. Quite often the individual is known to have an old localized rculous process at one apex but not uncommonly there has been no lence of ill health and the family history is good.

The onset is practically identical with that of lobar pneumonia. The ent, after exposure to cold, is suddenly seized with a chill, and severe in the side. The temperature rises rapidly and is continuous in (see Fig. 249). There is a suppressed cough, the sputum is blood- ed or even typically rusty in character and the respiratory rate is kened. No suspicion is entertained that the condition is other than bar pneumonia until the duration of the latter has been exceeded and risis occurs. In addition the temperature becomes hectic in type the sputum mucopurulent. Tubercle bacilli usually can be demon- ted at this time. The patient suffers from chills and sweating and emaciates rapidly. A fatal issue may occur in four or five weeks he condition may become subacute and be prolonged for months. If a limited portion of a lobe is involved the acute symptoms may ide and the lesion becomes arrested or slowly progressive.

Physical Signs.—The physical signs of acute pneumonic phthisis lways found at one or the other apex of the lung; never at the bases.

They differ in no particular from those occurring in lobar pneumonia, namely, increased tactile fremitus, dulness on percussion, exaggeration of the voice sounds, bronchial breathing and fine crackling râles. Some observers point out that the breath sounds in tuberculous pneumonia are more often suppressed and distant than in the croupous variety and that this is of diagnostic significance.

Later in the disease there may be evidences of breaking down of the lung at the apex. At first large moist râles are heard; later there may be signs of a cavity.

Diagnosis.—There is no doubt that the acute forms of tuberculosis are very frequently mistaken for *lobar pneumonia*. The acute onset, the high continuous fever, pain in the side, and blood-streaked sputum together with physical signs indicating partial or complete consolidation are responsible for this. In many such cases the acute process subsides in a few weeks and the patient apparently recovers; in others a tuberculosis is said to have developed after the pneumonia. It is to be remembered that tuberculosis very often undergoes an acute exacerbation lasting for a few weeks and again becomes quiescent. Of 4466 cases seen at the Phipps Institute no less than 17.77 per cent. gave a history of having had an attack of pneumonia. How many of these attacks were in reality an acute exacerbation of the tuberculosis is problematical, but it is fair to assume that a considerable number were of that nature.

While it is often impossible during the acute stage to differentiate between lobar pneumonia and pneumonic phthisis the following points are helpful: (1) A history of tuberculosis in the family; (2) a knowledge of the existence of an old tuberculous lesion; (3) a longer duration in the case of pneumonic phthisis; (4) herpes are common in croupous pneumonia and rare in the tuberculous form; (5) a leukocytosis is common in lobar pneumonia; and (6) the location of the lesion. Croupous pneumonia most frequently involves the lower lobes but may occur in the upper lobes. As a rule acute tuberculosis is limited to or most marked in the upper lobes. If, however, there is a lesion already in the upper lobe a rapid invasion of the lower lobe may take place with symptoms and physical signs similar to those of lobar pneumonia. The latter group offer the greatest difficulty and it is, as a rule, not possible to tell which disease is present until several weeks have elapsed. If the symptoms and physical signs persist it can be assumed with certainty that the lesion is tuberculous in nature. When the upper lobes are involved, tuberculosis should always be kept in mind, especially if there is a family history of tuberculosis or the patient gives a history of having been in poor health (see also p. 423).

FIBROID PHTHISIS

In certain instances tuberculosis assumes a very chronic form characterized by extensive fibroid changes in the affected lung. The arrest of a tuberculous process is largely due to the formation of fibrous tissue about the lesions and even in those cases in which the disease progresses the rapidity of its advance is determined by the amount of fibrosis which takes place. In the acute types of pulmonary tuberculosis in which the course of the disease is relatively short and the pulmonary lesions widespread, there is little or no evidence of fibroid changes. The opposite extreme is encountered in the type of the disease under discussion.

Here the formation of fibroid tissue far exceeds what is needed, thus giving rise to pulmonary fibrosis with its results.

Clarke, Hadley and Chaplin in their authoritative monograph on the subject of Fibroid Phthisis distinguished three forms: (1) Tuberculo-fibroid disease, a condition which is primarily tuberculous but in which there is an overgrowth of fibrous tissue; (2) fibro-tuberculous disease, a condition which is primarily a pure fibrosis but later becomes tuberculous; and (3) pure fibroid disease of the lung.

The first type may from the beginning be characterized by an excessive amount of fibrous tissue or the chronic ulcerative type of tuberculosis may change into the fibroid type. In the latter instance a single isolated cavity at the apex may be surrounded by a thick, dense wall of fibroid tissue involving both the surrounding pulmonary tissue and the pleura. In a case seen at the Phipps Institute a large apical cavity was surrounded by dense cartilaginous material which in places was over an inch in thickness. The fibrous process may be limited to the apex or gradually involve the entire lung.

In regard to the fibro-tuberculous and pure fibroid types it has been objected that they are only examples of "phthisis of which the history has been forgotten." Pure fibrosis of the lung, although rare, does, however, exist. Two excellent examples of the disease have been seen at the Phipps Institute. The symptoms and physical signs of fibroid phthisis differ in no particular from those encountered in pulmonary fibrosis or cirrhosis of the lung. The sections on Bronchiectasis and Pulmonary Fibrosis should be consulted in connection with this subject.

ACUTE MILIARY TUBERCULOSIS

This form of tuberculosis requires special consideration because of its anatomical relations and clinical manifestations. "The term 'miliary tuberculosis' is applied to the appearance of numerous small, gray or grayish-red tuberculous nodules about the size of a millet-seed and approximately at the same degree of development in an organ, especially the lung, or in only a certain portion of the lung. By general miliary tuberculosis we designate the fairly regular distribution of such nodules in all or several of the organs of the body, particularly the lung, liver, spleen, kidney, brain, bone-marrow, etc. As a rule, the disease is acute and characterized by a tumultuous course, terminating in death usually in a few weeks" (Cornet).

Etiology.—The modern conception of miliary tuberculosis dates from the statement made by Buhl (1856), that miliary tuberculosis is a specific infectious disease, the miliary nodule bearing the same relation to a caseous focus as the metastatic abscesses in pyemia have to the primary focus of suppuration. Some years later Weigert furnished the proof as to the manner in which the tubercle bacilli are disseminated. This may be brought about by direct invasion of the walls of blood-vessels or the thoracic duct or by the rupture into one of these vessels of an adjacent tuberculous focus. Miliary tuberculosis occurring under natural conditions is analogous to experimental tuberculosis in which an enormous number of tubercle bacilli are injected directly into the vein of an animal. In both instances the body is suddenly overwhelmed. When the lungs are specially affected it is generally the result of a pulmonary lesion or a caseous tracheal or bronchial lymph node, which has broken down and discharged its contents into a branch of the pulmonary vein. In the more widely disseminated form of the disease the most frequent source of the infection is the thoracic duct. Longcope[1] in a study of 19 typical cases of generalized acute miliary tuberculosis found in 14 more or less extensive tuberculosis in the thoracic duct, usually with caseous nodules. Ullom[2] examined the thoracic duct in 17 cases of chronic pulmonary tuberculosis but found neither tubercles in the structure nor tubercle bacilli in the contents of the ducts.

Both the site of the initial lesion and the localization of the miliary nodules vary greatly in different cases, as can be seen from the following examples which have come to my notice. In one instance a widespread dissemination followed the rupture of a caseous lymph node into the thoracic duct in a case with old hip-joint disease; in another the miliary nodules were confined entirely to the abdominal organs, the primary source being a single tuberculous ulcer in the ileum; in another the nodules were limited to one lung the source of the infection being an old cavity surrounded by a dense cicatricial wall; in three instances tuberculosis of the testicles was followed by miliary tuberculosis, the dominant feature being localization in the meninges.

The relation which pulmonary tuberculosis bears to the acute miliary form of the disease is an interesting one. At first sight it would seem reasonable to believe that this would be the most frequent predisposing factor. As a matter of fact widespread dissemination is unusual and while miliary tubercles in parts of the lung previously free from disease are frequently found at autopsy their presence is often overlooked during life. It is of course recognized that in all cases of chronic pulmonary tuberculosis most of the organs of the body will show, either macroscopically or microscopically, small nodules, but which, in the strict sense of the term, are not miliary tubercles. They point to a hematogenous dissemination shortly before death. These nodules are possibly due in part to dead tubercle bacilli which are known to produce such changes. Histologically these nodules are evidently of recent formation. In a study of 37 livers from phthisical subjects Ullom[3] found small tuberculous foci present in 30 or 81 per cent. Walsh[4] obtained similar results in a study of the kidneys.

Although generalized miliary tuberculosis is comparatively infrequent as a terminal manifestation of pulmonary tuberculosis, miliary tuberculosis limited to the lungs or the meningeal type of the disease are not uncommon. Miliary tuberculosis of the lungs alone is most apt to develop in cases with an obsolete quiescent lesion, especially if this lesion is caseous. The danger is far less if the lesion has become encapsulated with fibrous tissue. The meningeal type of miliary tuberculosis is occasionally the terminal manifestation of phthisis. Brown[5] states that tuberculous meningitis was the ultimate cause of death in 8 of 352 males (2.27 per cent.) and in 3 of 322 females (0.93 per cent.) discharged from the Adirondack Cottage Sanatorium.

Miliary tuberculosis may occur at any age period but is far more common in early life than later. Of the exciting causes the acute infectious diseases are the most important, especially measles and whooping cough. Typhoid fever is often cited as a predisposing cause but in the vast majority of such cases there has been an error in diagnosis. As will be shown later, enteric fever and the typhoid form of miliary tuberculosis are differentiated only with the greatest difficulty. Acute dissemination is not infrequent following an operation on a tuberculous bone or joint. Operations on individuals with frank pulmonary tuberculosis are rarely followed by miliary tuberculosis. There may be, however, an exacerbation of the disease. In such cases the dissemination in the lungs is peribronchial in character and is due to the insufflation of tuberculous material. If this occurs it is practically always due to ether; other forms of anesthesia are well borne by phthisical subjects. Among other predisposing causes may be mentioned traumatic injuries, pregnancy, chronic debilitating affections, or any factor which tends to reduce the vitality, such as grief or worry. Occasionally miliary tuberculosis follows the rapid absorption of a pleural exudate.

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[1] *Bulletin Ayer Clinical Laboratory*, Pa. Hospital, No. 3, 1906.

[2] 5th Annual Report, Phipps Institute, 1908.

[3] 2d Annual Report, Phipps Institution, 1905.

[4] 3d Annual Report, Phipps Institute, 1906.

[5] Osler's "Modern Medicine," vol. iii, 1st ed., p. 302.

It is difficult to estimate the frequency of this type of tuberculosis. It is encountered far more often in general hospitals than in institutions which care exclusively for tuberculous patients.

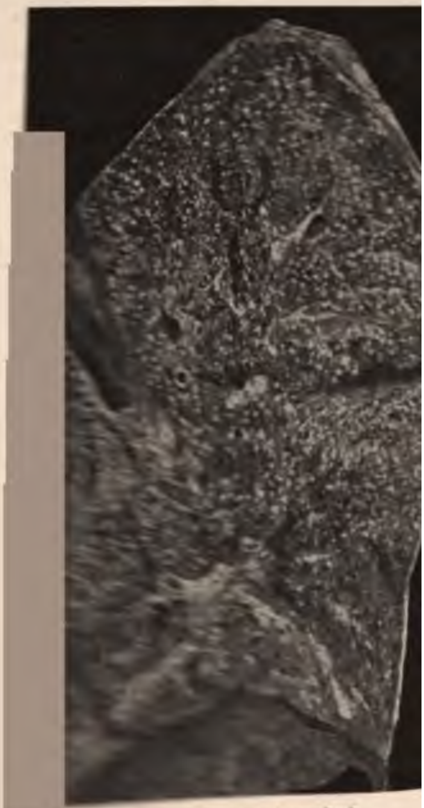
Morbid Anatomy.—Every case of miliary tuberculosis, whether widely disseminated or limited to a single organ, has its origin in an old caseous focus. At autopsy this may be easily demonstrated or it may be so small or so situated in some portion of the body not routinely examined, as to escape detection. Anatomically the disease is characterized by the presence of countless miliary nodules varying in size from that of a pinhead to a millet-seed (Fig. 267). The tubercles may be confined or at least most marked, in a single organ or they may invade nearly every portion of the body. The organs which are especially vulnerable are the lungs, liver, spleen, and kidneys, the serous membranes and often the choroid. For some reason the pancreas, the skeletal muscles and the lymph nodes are very resistant and rarely show the presence of tubercles.

The predominance of tubercles in certain organs will often give the clue as to the site of the original focus. Thus if the liver is most involved the primary lesion is in the area drained by the portal vein; if the tubercles are most numerous in the spleen, liver and kidney the infection has probably been carried by the pulmonary veins; uniform distribution throughout both lungs points to the thoracic duct or one of the systemic veins. In the pulmonary type the demonstration of an old lesion in the lungs is usually an easy matter.

The most recent nodules consist of minute grayish, semitransparent points. If several eruptions have taken place, the tubercles will show variations in size and color. The older nodules are larger with greyish-yellow, yellowish-white centers. Microscopically many minute tubercles may be demonstrated which cannot be recognized by the naked eye. The affected organs are usually increased in size.

The lungs are larger than normal and intensely hyperemic. On section the cut surface appears dark red and granular and the nodules feel hard. When the condition is secondary to chronic pulmonary tuberculosis of the lungs in adults the nodules are less sharply defined and tend to coalesce. The nodules are always most numerous in the immediate neighborhood of the original focus and tend to diminish in number as the base of the lung is reached. In children, on th monary nodules are discrete and thickly and u throughout the lungs.

The bronchi are inflamed and in children es lymph nodes are enlarged and usually caseous. extensively infiltrated with discrete miliary granula is widespread the peritoneum and meninges are tubercles. The presence of choroidal tubercles interest.



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cases studied by Longcope[1] the process was subacute or chronic. tubercles or caseous nodules, were scattered in small numbers gh the various organs and during life the course of the disease was acted, lasting from three to nine months.

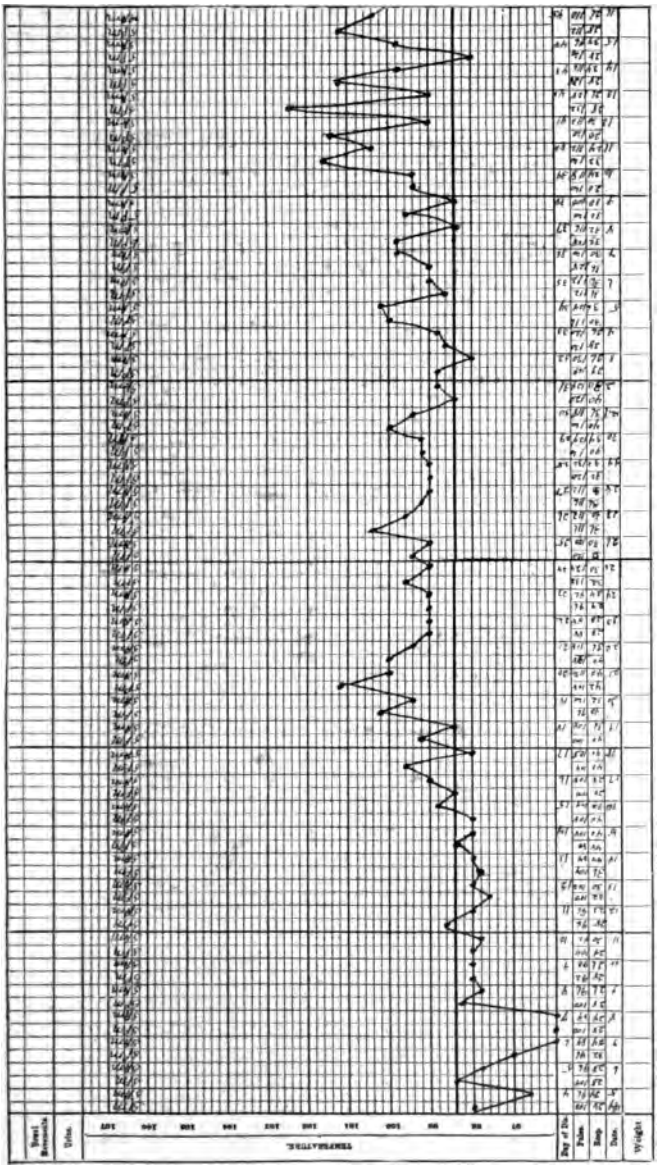

FIG. 268.—Miliary tuberculosis.

inically the cases are usually grouped as follows: (1) the typhoid in which the picture is that of an acute general infection; (2) the nary form; and (3) the meningeal form. It is to be understood that this classification is somewhat arbitrary and that in some instanc the typhoid form will show in the terminal stage evidences of localizatio in the lungs or the meninges or both.

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[1] *Loc. cit.*

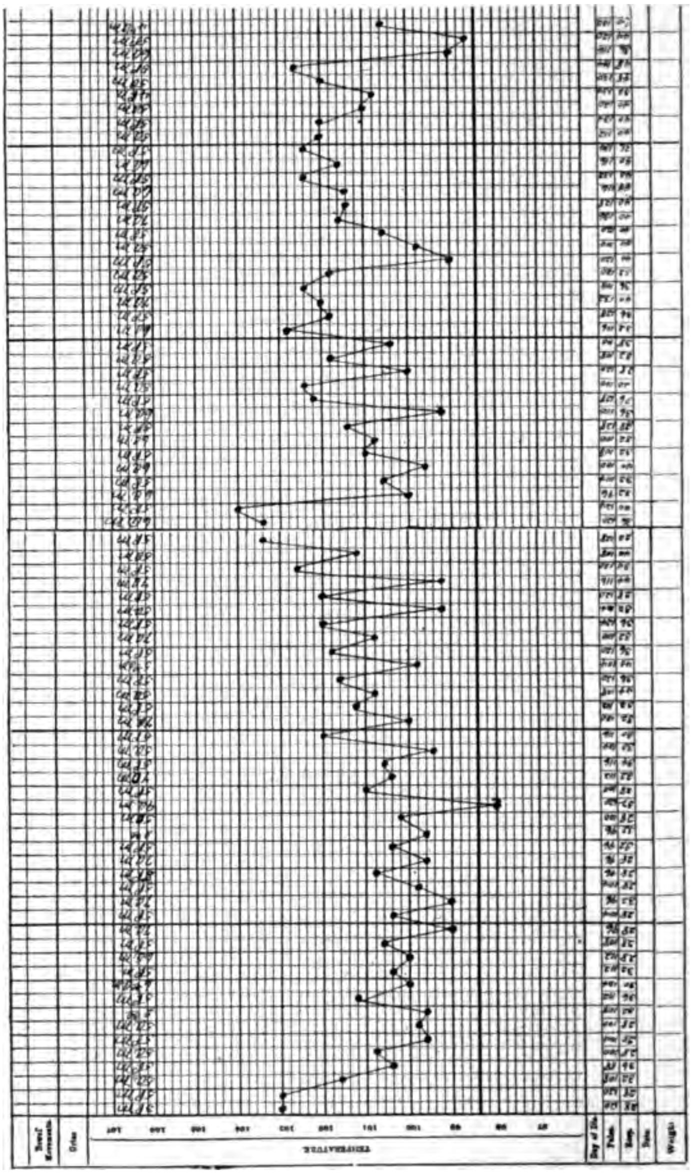

FIG. 269.—Miliary tuberculosis.

THE TYPHOID FORM

The onset is usually insidious and the history is often almost identica with that obtained in cases of typhoid fever. There is an initial perio which the patient complains of malaise, headache, and anorexia. igestive disturbances are often present and there may be some cough. he temperature is elevated and for some days may closely resemble the pe of fever seen in typhoid cases. In the great majority of cases, hower, the temperature is characterized by its irregularity. The remissions e much more marked than in typhoid fever usually amounting to two three degrees. In some instances the temperature is remittent and casionally there is little or no fever. If the temperature chart is carelly examined it will often be found that the temperature is inverted m time to time, that is the daily rise in the fever occurs in the morning d the drop in the evening. The pulse is rapid and weak but seldom crotic. An increase in the respiratory rate is usually present. As the sease progresses the tongue becomes dry and the lips and teeth are vered with sordes. Emaciation is usually marked. Nervous sympms are often prominent and consist of restlessness, delirium, subsultus, perasthesia and a tendency to mental torpor and coma. Meteorism frequently present and there may be diarrhea. Occasionally blood is esent in the stools. The general appearance of the patient and the mptoms are essentially the same as those occurring in cases of toxemia m any cause.

The physical examination may furnish but little information in the ginning or indeed throughout the course of the disease. The heart unds are rapid and weak and the first sound may closely resemble the cond. The lungs may show no abnormality or the presence of a more less diffuse bronchitis. A marked increase in the respiratory rate ould arouse one's suspicions as to the presence of pulmonary mischief. heyne-Stokes breathing is occasionally present in the late stages of the sease. Slight enlargement of the spleen can often be made out. he presence of choroidal tubercles is often cited as an important diagstic sign. They should always be looked for in suspected cases of iliary tuberculosis but it is only rarely that their detection confirms e diagnosis. Very often in miliary tuberculosis small reddish and ghtly elevated spots appear on the skin. Although they have a superial resemblance to the rose spots seen in typhoid fever they do not come it in successive crops and in addition, they lack the other characteristics the true typhoid eruption. A petechial eruption, appearing especially out the wrists often occurs in the late stages. Rarely jaundice occurs there is extensive involvement of the liver. I have seen one such case. he urine may show traces of albumen. Ehrlich's diazo-reaction is of tle value as it occurs both in typhoid fever and tuberculosis. The ukocyte count is normal or slightly increased. Toward the end pulonary or meningeal signs and symptoms or both may be marked. In e majority of cases, however, localizing signs are absent and the tient finally dies with all the evidences of a general toxemia.

Diagnosis.—Considering the widespread distributions of the disease d the pronounced character of the symptoms it is remarkable how freuently the condition is mistaken for typhoid fever or malignant endorditis. It is notorious that miliary tuberculosis is far more often misken for *typhoid fever* than the reverse. The confusion is greatest in ses in which there is no obvious tuberculous focus. Symptoms of tyoid fever developing in an individual with old hip-joint disease or other onic tuberculous lesion, should always suggest miliary tuberculosis.

In typhoid fever the temperature curve is characterized by a steady ascent followed by a period of slight remissions and ~~later by intermissions~~. In both miliary tuberculosis and *malignant endocarditis* the temperature is very irregular and does not conform to any definite type. Eruptions occur in all three conditions. That of typhoid fever consists of the so-called "rose spots" which most often appear over the abdomen and lower chest. A petechial eruption may be present in both miliary tuberculosis and malignant endocarditis. This eruption may appear on any portion of the body but in miliary tuberculosis it shows a preference for the wrists. The presence of a cardiac murmur associated with symptoms of a general toxemia should always suggest malignant endocarditis; unfortunately in not a few of these cases examination of the heart gives no clue as to the true nature of the trouble.

Modern laboratory methods offer the most certain means of differentiating these conditions. Blood cultures will show the presence, usually of a streptococcus, in cases of malignant endocarditis. In typhoid fever Eberth's bacillus can often be demonstrated during the early days of the disease. The Widal reaction is looked upon as strong evidence in favor of typhoid fever but in several instances which have come to my notice a positive Widal was obtained in cases which at autopsy proved to be general miliary tuberculosis.

PULMONARY FORM

In this form the brunt of the infection is borne by the lungs and the symptoms and physical signs are more or less distinctly pulmonary in character. The onset may be gradual or sudden, usually the former. In the majority of instances when the disease occurs in adults the patient is known to have a chronic pulmonary lesion or to have been subject to a chronic cough. In children it is most apt to succeed one of the acute infections, particularly measles, or whooping cough. The patient complains of malaise, headache and anorexia. Cough and expectoration are always present. The cough may, for a time be hacking in character and unproductive but in most cases it i accompanied by mucopurulent expectoration. The sputum is ofte blood-streaked and occasionally the presence of an old cavity or mor or less dense infiltration. The signs of the old lesion may consist o nothing more than slight retraction and diminution of expansion an suppression of the respiratory sounds. Over the remainder of the lun the percussion note may be resonant or even hyperresonant owing t the small size of the miliary tubercles, the fact that they are scattere and also because there are often varying degrees of emphysema. I some instances patches of dulness may be elicited over areas in which th tubercles have coalesced in somewhat the same manner as occurs i tuberculous broncho-pneumonia. In children a broncho-pneumoni type of the disease is not unusual. On auscultation the breath sound may be harsh but more often they are obscured by râles. These ar numerous and in the beginning are intense and crackling in characte As the disease progresses large mucous and sibilant râles are also hear Over the patches of consolidation the breathing is bronchial or bronch vesicular in character. The heart sounds may be clear but often the are obscured by râles. The spleen is usually enlarged.

If an old pulmonary lesion is present tubercle bacilli can usually demonstrated in the sputum. In the absence of such a lesion tubercle bacilli are absent as death usually takes place before the miliary tubercles break down. The urine may show traces of albumen. The leukocyte count may be normal or slightly increased.

Diagnosis.—As has been previously pointed out miliary tuberculosis affecting a portion of the lung previously free from disease is not infrequent as a terminal event in the chronic pulmonary type of tuberculosis. As a rule its presence is unrecognized during life. When, however, it arises secondarily to a limited apical lesion or follows an attack of measles or whooping cough in children the diagnosis is relatively easy. The dyspnea, cyanosis, cough, and evidences of a severe infection should always arouse suspicion. This becomes still stronger with the finding of evidences of pulmonary damage at one of the apices. The presence of tubercle bacilli in the sputum furnishes the positive proof.

MENINGEAL FORM

(Tuberculous or basilar meningitis, acute hydrocephalus, "water on the brain.")

Etiology.—Miliary tuberculosis characterized by meningeal symptoms is by far the commonest form of the disease. It occurs more often in children than adults. Of 8877 deaths from tuberculosis of all kinds in children, under 5 years, 3347 were due to meningitis (Cobbett). Tuberculous meningitis is occasionally the terminal event in cases of chronic pulmonary tuberculosis. The condition is practically always secondary to a tuberculous lesion elsewhere in the body. In the few instances in which it is apparently primary it is probable that the search for the initial lesion has not been sufficiently thorough. The primary lesion may be located in portions of the body which are omitted in a routine autopsy, as for instance, the middle ear or the bones.

Morbid Anatomy.—Invasion of the meninges is practically always through the circulation being secondary therefore to a lesion elsewhere in the body. The most common primary foci are to be found in the lungs or lymph nodes. Rarely the meninges are invaded by extension from adjacent structures. The tubercle bacilli being deposited in the meninges produce small tubercles. These may be few in number and limited to the base of the brain or they may be quite widely scattered. Often they are found along the Sylvian fissure. The condition is usually bilateral but one side may be more extensively involved than the other. The tubercles consist of small whitish or grayish-white nodules the size of a pinhead or smaller, distributed along the course of the blood-vessels. In most cases the tubercles are associated with a turbid, seropurulent exudate which is most marked at the base of the brain. It may extend, however, up over the lateral hemisphere of the brain especially along the Sylvian fissure. As a result the basal convolutions may be flattened and the cortex swollen and edematous. The pia-arachnoid is usually congested and edematous. The lateral ventricles are dilated and contain a turbid fluid similar to the exudation elsewhere. Occasionally the process extends downward involving the upper part of the spinal cord.

A chronic form is not uncommon in which a single tuberculous nodule is formed as the result of the coalescence of a small cluster of tubercles. This type may be more or less latent or give rise to symptoms similar to those encountered in cases of brain tumor.

Symptoms.—As the symptoms are dependent on irritation of the nerves they will vary considerably in different cases according to the portion of the central nervous system most involved. The onset is rarely sudden. In children there is apt to be a history of failing health for a few weeks or the child has but recently recovered from an attack of measles or whooping cough. In adults all the cases of tuberculous meningitis which I have seen have been associated with an obvious primary lesion, usually in the lungs; in three instances there was a tuberculous testicle also. The disease may be ushered in suddenly with a convulsion but headache is usually the first symptom which attracts attention. The headache may be the only symptom for weeks and in several cases which I have seen an almost constant headache preceded the meningitis for over two months. Associated with this symptom, in the majority of cases, are retraction of the neck, vomiting, constipation and fever. In children the headache may manifest itself by the child putting its hand to its head and if the pain is severe the child may at the same time cry or scream. The vomiting is often explosive in character, occurs without apparent cause and is independent of the taking of food. The fever is not marked in the beginning but gradually becomes higher. The pulse at first rapid may become slow; when present bradycardia is of great diagnostic importance, but this symptom is more often absent than present. In the last ten cases I have seen in adults slowing of the pulse rate occurred in but two. Later the abdomen becomes retracted and symptoms indicative of irritation of the nerve centers appear. Eye symptoms are common. These may be photophobia, irregularity of the pupils and paresis or paralysis of the ocular muscles. Facial paralyses are also seen at times and Kernig's sign can usually be elicited. In the final stage there is unconsciousness and convulsions may occur. The pulse again becomes rapid and the temperature falls to normal or subnormal.

In an individual who is known to be tuberculous and in whom there develops severe headache, vomiting, fever, bradycardia and signs of irritation of the nerve centers, a diagnosis of tuberculous meningitis can be made with reasonable certainty. In children in whom it may be difficult to establish the presence of a primary focus, spinal puncture should always be resorted to. The fluid withdrawn is usually clear but may be slightly turbid and under high pressure. The cellular elements are moderately increased. In the very early stages polynuclear cells may predominate but later there is usually a marked lymphocytosis. In the majority of cases tubercle bacilli can be demonstrated in the fluid although in some cases prolonged and patient search is required.

The duration of the disease is, as a rule, from two to four weeks. Occasionally fulminating cases are seen in which death takes place in a few days; in other instances the disease is quite prolonged and chronic. The accepted opinion is that tuberculous meningitis is always fatal and although several cases of recovery have been reported many question the reliability of the observations.

MYCOTIC INFECTIONS OF THE LUNGS

The pulmonary symptoms produced by mycotic infections of the lungs are, in many instances, not to be distinguished from those occurring in tuberculosis. Furthermore, the physical signs are apt to be identical licate a localized process at the apex, a widespread miliary process, ronic type of disease with consolidation or cavity formation. In f their great similarity to tuberculosis in both symptoms and al signs, it is impossible to tell how frequent these infections really s those actually recorded in the literature are comparatively number. It is a reasonable assumption, however, that many of diagnosis do occur. It is quite likely also that there are not a stances in which the true nature of the trouble has escaped notice n the autopsy room, inasmuch as the small nodules could readily en for miliary tubercles. This error could very easily be made in nens of blastomycosis of the lungs that I have seen. In cases of ioidal granuloma the resemblance is even more pronounced.

iere are two very common errors made in determining the character lmonary infections. The first is the readiness with which many tioners accept a single negative sputum examination as evidence uberculosis can be ruled out. It must be borne in mind that one or a number of negative examinations are of no value in excluding tuberculosis, as the great majority of cases of true incipient tuber- s do not show tubercle bacilli in the sputum.

ie second error is in assuming that because the patient presents evidence of widespread pulmonary disease and his symptoms are tive of that condition that there can be no doubt of the diagnosis. n cases of this class that some of the unusual types of pulmonary ion escape recognition. Either a sputum examination is not lered worth while, or the occurrence of a number of negative ex- itions does not arouse the suspicion that some organism other than ibercle bacillus, may be the true cause of the trouble.

cannot be emphasized too strongly that in every case in which the toms and physical signs point to tuberculosis, and yet repeated nations of the sputum fail to reveal tubercle bacilli, some other ing agent should be thought of.

PULMONARY STREPTOTHRICOSIS

iere is an extensive literature on streptothricosis which is more s confusing to the student by reason of the fact that the disease has described under such a variety of names. The following terms have employed to designate this group of organisms: Actinomyces, thrix, nocardia, oöspora, discomyces, pseudo-tubercle bacillus, etc. the appearance of the papers by Musgrave, Clegg, and Polk[1] 'oulerton,[2] the term streptothrix adopted by these authorities, has into general use. It is to be understood that the term is not ap- to a single organism but to a group consisting of a number of dis- species. While many writers describe the actinomyces as one of pecies of the streptothrix group, it seems preferable to give it a ate place. This is justified not only because of certain distinct ral differences but also because of its pathological and clinical 'estations.

inically, the streptothrix group is the most important of the tic organisms. Not only is it by far the most common of these ions but in addition it is readily confused with tuberculosis. There

[1] *Philippine Jour. Sc.*, 1908, vol. iii, 447.

[2] *Lancet*, Feb. 26; March 5 and 19, 1910.

is good reason to believe that streptothricosis is much more prevalent than the reported cases indicate.

This group of organisms is in need of more extended study, especially with a view of determining their pathogenicity. Their presence in the sputum does not mean necessarily, that they are responsible for the symptoms presented by the patient. Up to date some thirty cases have been reported in which a streptothrix organism had produced definite pulmonary lesions and there can be no doubt but that certain members of the group possess definite pathogenic properties. On the other hand it is to be borne in mind that streptothrix organisms very frequently are found in the sputum either alone or in association with the tubercle bacillus or other organisms without its being apparent that they have any significance. During the past winter, at the White Haven Sanatorium, I had the sputum of a number of patients examined with the view of determining whether those free from tubercle bacilli might not be suffering from some other infection. Of 24 cases presenting the symptoms and physical signs of an early tuberculosis but without tubercle bacilli in the sputum 18 or 75 per cent. showed streptothrix organisms. The frequency with which the streptothrix was found seemed to be against its being the etiological factor. A second group comprising 36 cases, in which tubercle bacilli were found, also showed the presence of a streptothrix in 10 or 27.7 per cent. Every effort was made to eliminate contamination from the mouth, the teeth being cleaned and the mouth washed with a mild antiseptic in each instance. The streptothrix was found in each instance in small yellowish granules but never in pure culture, streptococci being present in addition.

In none of the cases studied by us was the streptothrix in the least acid fast. It is well known that the members of this group vary greatly in their ability to withstand acid; some are readily decolorized by the weakest acid solutions; others are resistant to everything except 30 per cent. nitric acid. It is quite possible that the degree to which they are acid fast may have some bearing on their capacity for mischief and that the more resistant they are to acid the more nearly they approach the tubercle bacillus.

My own experience has led me to the belief that non-acid fast streptothrix organisms are relatively common and that they are to be looked upon in the majority of instances, as having little pathological significance.

Etiology.—It is not known how the infection is acquired. Certain species of the group have a wide distribution in nature and have been found in the air, water, soil and on food stuffs. It may be assumed that these organisms gain entrance into the body in much the same way as the tubercle bacillus and that pulmonary infection takes place, for the most part, by inhalation and, to a lesser extent, by ingestion.

It is well known that streptothrix organisms can be found in the mouth, nasopharynx, crypts of the tonsils and in the material from dental caries. Lord[1] has expressed the belief that the tonsils and carious teeth are often harbingers for these fungi and that they may start up active disease elsewhere. Nearly one-half of the recorded cases involve the head and neck regions (Claypole). Clinically this is important as there is nothing characteristic in the appearance of the abscesses or enlarged ph nodes to differentiate the condition from tuberculosis. From the es so far recorded it is estimated that chest infections constitute about per cent. of all cases of streptothricosis.

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[1] *Jour. Am. Med. Assoc.*, 1910, lx, p. 1261.

Morbid Anatomy.—The distribution of the lesions in the lungs is y similar to that seen in tuberculosis. The infection may be limited the upper lobe or by the time the patient comes under observation lung and the apex of the opposite lung may be involved. As a rule, disease is slow and progressive and tends to remain localized but it y become acute and assume a pneumonic course. In the final stage infected area is markedly granulomatous, this tissue replacing all the cells and bronchioles. The nodules show little tendency to caseate d break down and cavity formation is unusual. In some instances, h as the case described by Flexner,[1] small nodules resembling miliary ercles are scattered through the lung. A marked feature of most es is the formation of fibroid tissue in the lung. This doubtless is gely responsible for the marked dyspnea which is characteristic of the uble in many cases.

Among other pulmonary lesions which have been ascribed to these anisms may be mentioned broncho-pneumonia, bronchiectasis, abscess d gangrene. The two latter conditions may be the sequels to a tonsil- omy, the organism being squeezed out of the crypts and insufflated o the air passages. In one case seen at the Phipps Institute the only lanation of an extensive unilateral pulmonary fibrosis seemed to be treptothrix organism. In several other instances in which the fibrosis s associated with bronchiectasis, streptothrix organisms were found ong an extensive bacterial flora; but in these cases it was not possible determine whether the streptothrix organism was a primary or a ondary invader. Fibrinous pleurisy and empyema occasionally cur. Perforation of the chest wall is rare in marked contrast to the quency with which this accident occurs in cases of infection with the tinomyces.

Metastatic lesions are not uncommon, especially in the brain.

Symptoms.—The extraordinary fidelity with which infection with this up of organisms may ape pulmonary tuberculosis is gradually becom- more and more apparent. Bridge[2] has reported 17 cases, in 12 of ich the persistent absence of tubercle bacilli was the only evidence ainst the diagnosis of tuberculosis. The symptoms and physical signs re identical with those encountered in the latter disease. In 5 cases h streptothrix organisms and tubercle bacilli were found.

In common with tuberculosis cough, mucopurulent sputum, fever l some acceleration of the pulse rate are present in all cases. Loss of ght occurs in most cases and may, in some instances, be marked. spnea may be the one symptom for which the patient seeks relief. some instances the sputum is foul-smelling as the result of an asso- ted bronchiectasis. A frank hemoptysis or the more or less constant sence of blood-streaked sputum is a common symptom. Chest pain the occurrence of a definite pleurisy is also encountered in a large portion of the cases.

Physical Signs.—There is nothing characteristic about the physical ns. As a rule, the disease is found in one of the upper lobes or when the patient is first seen all of one lung and the upper lobe on the opposite side may be diseased. The signs are those obtained in any infiltrating process, namely, restriction of motion, impairment of the percussion note, bronchovesicular breathing, which is often more or less suppressed, and exaggerated voice sounds and râles. Less commonly there may be cavity signs. Occasionally one or both bases of the lungs are diseased while the upper lobes are healthy; these should not be confused with tuberculosis.

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[1] *Trans. Assoc. Am. Phys.*, 1898, p. 31.

[2] *Jour. Am. Med. Assoc.*, 1911, vol. ii, p. 1501.

It can readily be seen that so far as the symptoms and physical signs are concerned a correct diagnosis is not possible without a sputum examination. If tubercle bacilli are persistently absent from the sputum of patients who present every other evidence of tuberculosis, a diagnosis of the latter condition is not tenable. Under these circumstances the sputum should be examined for the presence of one of the mycotic organisms.

Sputum Examination.—A 24-hour specimen should be searched for small yellowish granules about the size of a pinhead. These will be found in the purulent and not the watery portion of the sputum. These small granules should be stained with carbol-fuchsin and Gram stains. The stained specimen shows the organism as isolated, slender filaments or a loose network of filaments which may be stained uniformly or present a beaded appearance. The staining properties of this group vary considerably especially with reference to acid fastness; some are readily decolorized; others are moderately acid-fast; and still others are almost as resistant to acid as the tubercle bacillus. This is important to bear in mind as one who is possessed of but a superficial knowledge of bacteriology may readily mistake some of the short fragments for tubercle bacilli. This mistake may occur if Gabbet's stain is employed as the decolorizing agent. An instance of this has come to my notice and in one of Bridge's[1] cases a similar mistake apparently occurred. Streptothrix organisms are readily decolorized with 30 per cent. nitric acid. If the tonsils contain cheesy crypts, or dental caries is present, these sources of contamination should be eliminated.

Cutaneous Test.—Claypole[2] has differentiated a number of species and from glycerinated bouillon cultures prepared *streptotrichins.* She found that skin tests made in the same manner in which tuberculin is employed gave definite reactions in people with frank streptothrix infections. Both tuberculosis and streptothricosis may occur in the same individual.

Diagnosis.—This rests on excluding every other source of the trouble. At present it is doubtful if any but the acid fast streptothrix organisms can be considered as pathogenic.

PULMONARY ACTINOMYCOSIS

Etiology.—In certain parts of the world infection with the actinomyces is fairly frequent, namely, Germany, Russia and Austria. In the London hospitals[3] no less than 135 instances of the disease were encountered in the decade of 1902 to 1912. In this country, although isolated case reports appear from time to time, the disease may be said to be comparatively rare in man.

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[1] *Loc. cit.*

[2] *Trans. Nat. Assoc. for the Study and Prevention of Tuberculosis,* 1914

[3] *Proc. Royal Soc. Med.,* 1912–1913.

The disease is widespread among cattle, and is often referred to as g-jaw." It is occasionally met with in other domestic animals. re is no evidence, however, that infection in man is acquired either n the milk or meat of diseased cattle, and it is exceptional that infec- occurs in man from direct contact with a diseased animal. The ction is believed to be acquired in the majority of instances by those aged in the handling of straw and grain, and by those in close contact h vegetable products. Infection apparently is through the alimentary respiratory tracts, as it is in direct connection with these that the ease is usually encountered.

Statistical studies which have been made indicate that the infection rimarily a pulmonary one in from 15 to 18 per cent. of the reported es.

Morbid Anatomy.—Actinomycosis may manifest itself as a diffuse nchitis; a widespread miliary process, or as a result of coalescence of actinomycotic nodules, necrosis and cavity formation may occur.

The disease may involve either one or both lungs. The initial infec- n is believed by some to occur more frequently in one of the lower es, although in most instances the disease has become so widespread the time it is recognized that the primary focus cannot be determined.

The changes produced in the lungs consist of the formation of small lules not unlike tubercles. These nodules may coalesce forming sses of considerable size and resembling sarcoma. As the disease gresses, an area of necrosed cells appear about the nodules and about s area a round-celled proliferation occurs. Connective-tissue pro- ration is usually marked about the nodules. The tendency of the ease seems to be to spread by direct continuity of tissue and, unlike erculosis, to ignore anatomical boundaries, and cause adhesions by olving all and every structure with which it comes into contact; this respect it resembles malignant disease.

When the lung is the seat of the primary infection the pulmonary focus spread by continuity of tissue until it has reached the pleural surface. two surfaces of the pleural sac then become adherent, the disease eading across the adherent surfaces and finally rupturing through an rcostal space without the occurrence of an empyema, although the er is commonly believed to be the real source of the trouble. The cess may occur at almost any part of the chest wall. In like manner or all of the thoracic contents may be involved by the disease.

When the lung is the primary seat of the disease, the cutaneous lesion rs somewhere on the chest wall. It is to be borne in mind, however, pulmonary disease may be secondary to a lesion in other parts of body. More than half of the recorded cases show that the primary n involves the skin of the face and neck; it may appear also in the ctures within the mouth.

The cutaneous lesion is characteristic in appearance. The skin itself pparently infected only in a slight degree, except that it is punctuated and there by small ulcers which mark the sites of discharge of small cesses in the subcutaneous tissue. The surrounding skin is apt to ent a dusky appearance because of the changes in the underlying cutaneous tissue, and there is rarely any spreading of the ulceration. haps the most noticeable feature of the lesion is the curious linear kering which occurs in the area about the ulcer.

Symptoms.—In pulmonary actinomycosis the onset is gradual, the patient complaining of weakness and gradual loss of strength and weight. Sooner or later cough and expectoration develop. The latter is commonly mucopurulent in character and in many of the cases the small sulphur granules which are characteristic of the disease may be seen. Blood-streaked sputum and hemorrhage are of infrequent occurrence. The fever is irregular or hectic in type. With involvement of the pleura, pain is experienced, and this may be dull or aching in character or sharp and stabbing as in acute pleurisy.

As the disease progresses the chest wall becomes involved and later an abscess appears in one of the intercostal spaces. Because of the pain, the physical signs and the discharge of pus through an interspace, an empyema or caries of a rib is usually thought to be present.

Physical Signs.—A thorough *inspection is essential* because of the associated cutaneous lesion, the recognition of which is of the greatest aid in making a correct diagnosis.

Neither the percussion nor the auscultatory findings give any clue as to the true nature of the trouble.

The following case is a typical example of primary pulmonary actinomycosis observed in the Phipps Institute several years ago.

Case No. 5376. Male, aged ten. Family history, negative. Child was said to have had pneumonia followed by empyema which was drained when three years of age. Ill health ever since.

Symptoms.—Cough, yellowish-white expectoration, one hemoptysis, dyspnea and some chest pain. Patient died four weeks after admission to the hospital. During this period he had a high continuous type of temperature (103°F.) with slight daily remissions, very rapid heart action and extreme dyspnea. Five sputum examinations were negative for tubercle bacilli.

Physical Signs.—The patient presented an emaciated appearance with restriction of motion over both upper lobes. Marked dulness over both upper lobes and at right base (site of old empyema). Over the dull area the breathing was either bronchial or broncho-vesicular in character with many crepitant and medium-sized râles.

Both the symptoms and physical signs pointed to advanced pulmonary tuberculosis. The warning of five negative sputum examinations was not heeded. After the diagnosis of actinomycosis had been made at the autopsy a specimen of sputum, sent to the laboratory the day before the child's death, was examined and showed both the sulphur-like granules macroscopically and the stained organism microscopically.

Autopsy Findings.—Left lung, upper lobe grayish-yellow in color on section granular and markedly edematous with scattered nodules size of pinhead resembling an early stage of tubercle. Throughout the lower lobe are numerous shallow cavities filled with small bodies of the size and color of actinomycotic granules.

The bronchi contains granular material.

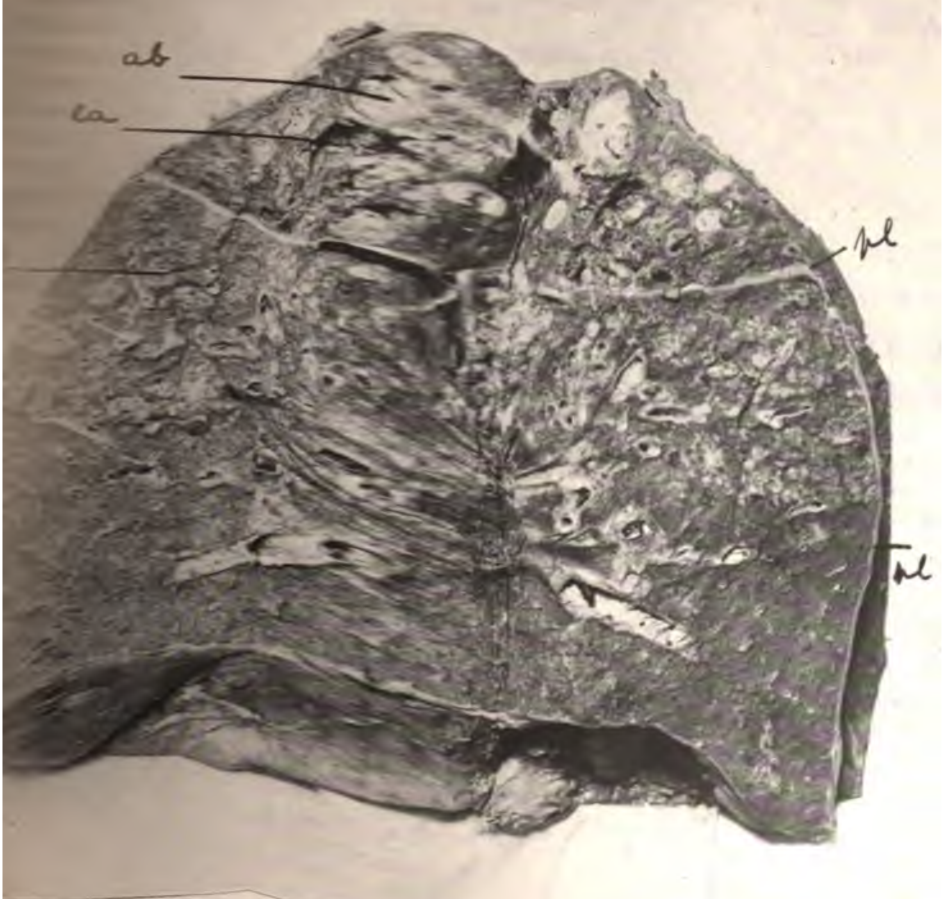
Right lung was firmly bound down by dense pleuritic adhesions. The upper lobe was yellowish in color and showed early gangrenous changes. On section dirty grayish-yellow pus exuded. The middle lobe contained several shallow cavities on the external surface containing granular material resembling actinomycotic granules.

The lower lobe showed no evidence of empyema. There were some isolated bodies resembling early tubercles.

The bronchial glands were enlarged and caseous. Histologically sections from the lungs showed edema, areas of broncho-pneumonia and rounded bodies having the appearance of actinomycotic granules. The tissue about these granules was necrotic and beyond the necrotic areas there was cellular infiltration. There was no evidence of tuberculosis.

Diagnosis.—Pulmonary actinomycosis is to be distinguished from tuberculosis, chronic bronchitis, empyema and caries of a rib. Unless the lesion is localized at the base of the lung the physical signs taken in conjunction with the symptoms will almost certainly lead to a diagnosis

The sputum is purulent or mucopurulent and often blooc
Occasionally a frank hemorrhage occurs. Owing to th
volvement, chest pain is common either in the form of a du
sation, or occasionally a sharp stabbing pain.
terminal stages of the disease emaciation may be very markec
however, the pulmonary lesions are found post-mortem to b



G. 271.—Right lung. Case 23. *ca*, Bronchiectatic cavity in the fibroid upper lob
bscesses whose contents were inspissated and surrounded by a wall of connectiv
e; *no*, blastomycotic nodules in the lower lobe, which from their distribution seem t
ronchogenic in origin; *pl*, thickened pleura. (*Boughton and Clark.*)

more extensive than either the symptoms or physical signs indicated
his is probably due to the fact that in some cases there is little or n
nflammatory reaction about the blastomycotic nodules.

Physical Signs.—A thorough inspection is of the utmost importance
This may reveal the presence of a swollen and painful joint, or a cutaneou
lesion.

inflammatory changes, or they may become confluent, forming larg areas with a necrotic center, which may break down. In the more chron types there may be extensive fibroid changes and dilatation of th bronchi.

The *skin lesions* may appear in the form of subcutaneous abscess or pustules; the latter are usually multiple and may occur successivel or in crops. The lesion may primarily take the form of an ulcer, or th ulcer may develop in the site of a ruptured abscess. The ulcer is "usuall surrounded by an areola of inflammation; the margins are raised an irregular, and the base is soft and covered with granulation tissue, whic may become fungoid and approach the surface, later assuming a papill matous appearance, which is more or less characteristic of the lesions chronic blastomycosis of the skin" (Fig. 270). Furthermore, as t lesion spreads peripherally it tends to heal in the center.

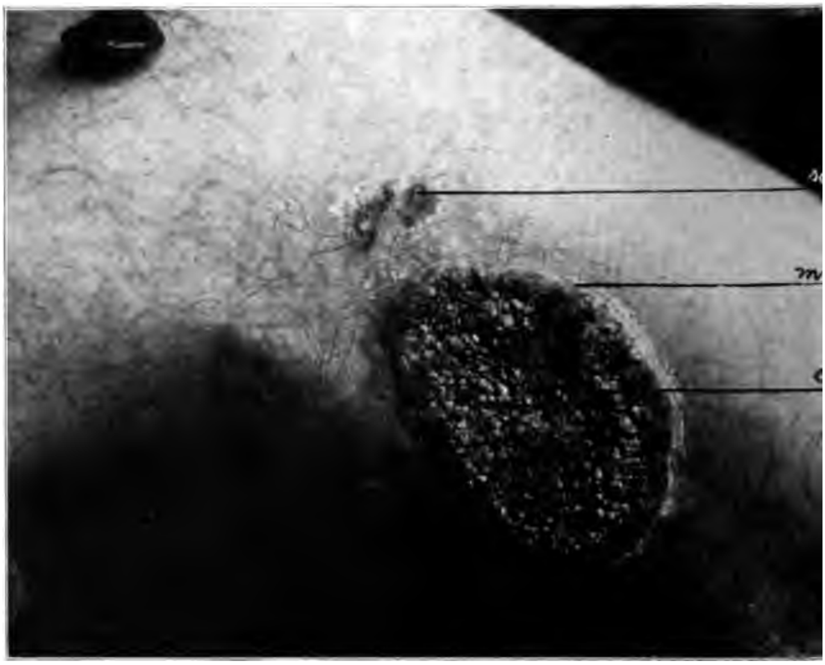


FIG. 270.—A typical lesion of cutaneous blastomycosis. *c*, **Papillomatous or v** rucose surface; *m*, raised indurated border containing **miliary abscesses; *sc*, scar of** lesion. (*By Courtesy of Dr. O. S. Ormsby.*)

Symptoms.—In systemic blastomycosis the initial symptoms m be hardly noticeable; more commonly they are those of an acute infe tion of the respiratory tract. This may resemble an ordinary "cold in the severer cases there is pain in the chest, fever, dyspnea, cough a the expectoration of blood-streaked mucopurulent material. Sooner later the cutaneous lesions appear.

As the disease progresses there is a gradual loss of weight and strengt accompanied with malaise, fever, irregular in character, but usual showing an afternoon rise; cough is usually present, and often severe character. The sputum is purulent or mucopurulent and often blood-streaked. Occasionally a frank hemorrhage occurs. Owing to the pleural involvement, chest pain is common either in the form of a dull aching sensation, or occasionally a sharp stabbing pain.

In the terminal stages of the disease emaciation may be very marked. At times, however, the pulmonary lesions are found post-mortem to be far more extensive than either the symptoms or physical signs indicated. This is probably due to the fact that in some cases there is little or no inflammatory reaction about the blastomycotic nodules.

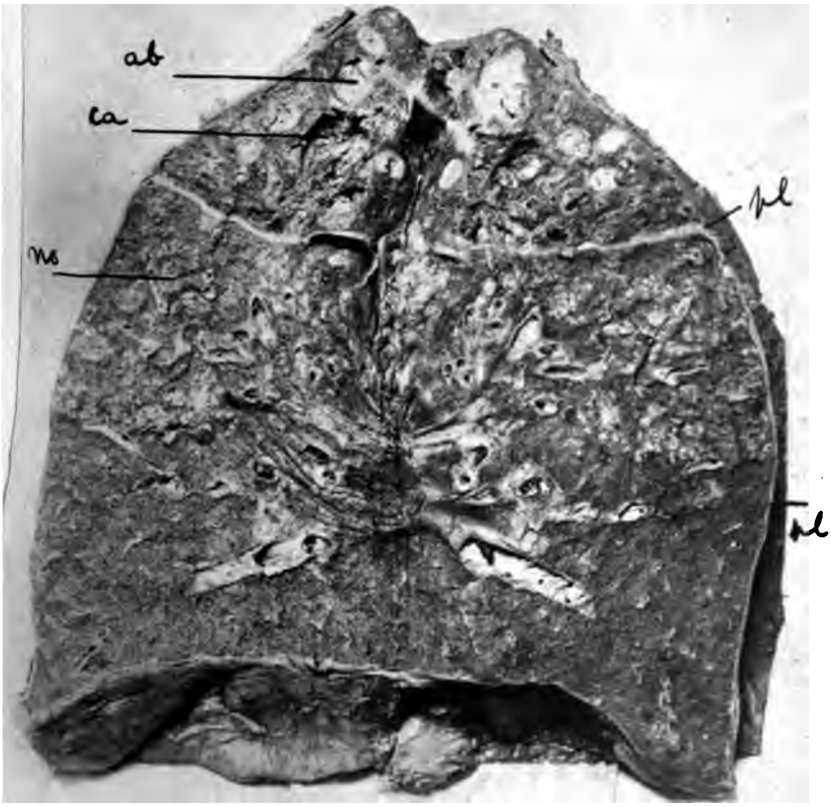


Fig. 271.—**Right lung, Case 23.** *ca*, Bronchiectatic cavity in the fibroid upper lobe; *ab*, abscesses whose contents were inspissated and surrounded by a wall of connective tissue; *no*, blastomycotic nodules in the lower lobe, which from their distribution seem to be bronchogenic in origin; *pl*, thickened pleura. (*Boughton and Clark.*)

Physical Signs.—A thorough inspection is of the utmost importance. This may reveal the presence of a swollen and painful joint, or a cutaneous lesion.

Restriction of expansion, flattening and other signs indicative of pulmonary damage are not peculiar to this disease. And the same is to be said of the percussion and auscultatory findings which are usually identical with those encountered in pulmonary tuberculosis.

The following case was seen at the Phipps Institute during the past year. Case No. 14912. A young Indian girl, aged eighteen, was referred to the wards of the Institute as a case of tuberculosis. She had complained of feeling badly for nearly a year. Six weeks prior to her admission she developed a cough and slight expectoration, mucopurulent in character. She had lost weight and had a slight rise in temperature every afternoon. She was also subject to paroxysmal attacks of dyspnea.

Physical examination showed nothing abnormal in the upper part of the lungs. Over both bases there was restricted motion, dulness on percussion, and feeble breath sounds and fine râles. Near the angle of the left scapula there were marked pectoriloquy and bronchial breathing. X-ray examination confirmed the clinical findings (see Fig. 372). Owing to the location of the lesions and the absence of tubercle bacilli the sputum was more thoroughly studied by my colleague, Paul A. Lewis. He found that the sputum contained the blastomycotic organism in pure culture and that other tests confirmed this finding.

Diagnosis.—Both the symptomatology and the physical signs of pulmonary or systemic blastomycosis often resemble tuberculosis so closely that a differentiation between the two is impossible without a sputum examination. Fig. 271 illustrates how easily a mistaken diagnosis of tuberculosis could be made.

As we have pointed out already, given a case which does not show tubercle bacilli, after a number of examinations, some other cause of the trouble must be sought for.

COCCIDIOIDAL GRANULOMA (California Disease)

Until within the past few years this disease was confused with blastomycosis. Recent studies, among which may be mentioned those of MacNeal and Taylor[1] and Brown and Cummins[2] have shown clearly that although the two diseases resemble each other, a differentiation may be made both from the clinical picture and the morphological and cultural characteristics of the infecting organisms.

Etiology.—The cause of coccidioidal granuloma is the *coccidioides immitis*, the true nature of which was first discovered by Ophüls and later by Wolbach. Prior to this it was thought the disease was due to a protozoon. These observers showed that the infecting organism belonged to the yeast group and was closely related to the blastomyces. The two organisms are differentiable by the endo-sporulation in the coccidioides and the budding in the blastomyces.

With but two or three exceptions all of the cases of coccidioidal granuloma so far reported, have occurred in the San Joaquin Valley, California. Inasmuch as there is no evidence of direct association of one case with a preëxisting case the presumption is strong that there must be some important local factor in causation. Recently Lipsitz, Lawson and Fessenden[3] have reported the case of a negro who lived in Missouri and had never been in California. Most of the patients have belonged to the class of day laborers. So far but one female has been reported as suffering from the disease.

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[1] *Jour. Med. Res.*, July, 1914.
[2] *Arch. Int. Med.*, April, 1915.
[3] *Jour. Am. Med. Assoc.*, April 29, 1916.

Since the disease was first described twenty-one years ago about 40 cases have been recognized.[1] Of recent years the number of cases has increased. This is probably due to the fact that physicians are becoming more familiar with the disease.

Morbid Anatomy.—The initial lesion may occur in the skin of the hands or feet, especially the former. If seen in its early stages the initial lesion takes the form of a papule or nodule which tends to form an ulcer very quickly. It is in the ulcerated state that the lesion is usually first seen. The appearance of the ulcer has been compared to a variety of conditions. MacNeal and Taylor state that the ulcers sometimes resemble mycosis fungoides. P. K. Brown[2] has likened them to the crusty lesions of pellagra; later they break down and resemble syphilitic or tuberculous ulcers. In place of the skin lesions painless subcutaneous abscesses are sometimes seen. The disease may also attack the bones, producing caries, or involve the joints giving rise to a form of arthritis which may be mistaken for tuberculosis. Meningitis due to the coccidioides has been noted also.

In most instances the primary infection is in the lungs although later skin ulcers or subcutaneous abscesses may form. In the lungs the lesions are those of a disseminated miliary tuberculosis. The nodules are grayish in color and in places coalesce. Small cavities are sometimes formed. Grossly the nodules cannot be distinguished from tubercles. Hektoen[3] states: "This disease presents the best mimicry of tuberculosis ever seen and the lesions cannot be distinguished from tubercles by the microscopic anatomy."

An excess of fluid in the pleural cavity is occasionally present. Coccidioidal disease differs from blastomycosis in that it shows a greater predilection for the lymphatic system, the skin lesions show a greater tendency to ulcerate, and in systemic cases recovery is unknown.

Symptoms.—It is emphasized by all who have had experience with coccidioidal granuloma that the symptoms, the clinical course and the pathological picture is often indistinguishable from tuberculosis.

As already stated the initial manifestation may be a skin lesion, a painless subcutaneous abscess, or an arthritis. Whether the lungs are involved primarily or secondarily, the symptoms and course of the disease differ in no particular from those occurring in tuberculosis. The patient loses weight, tires easily, has a cough, mucopurulent sputum, which is often blood-streaked, and afternoon fever. As the disease progresses the patient suffers from night sweats, emaciates and becomes anemic. Rarely the disease remains localized but early involvement of the regional lymph nodes and dissemination is the general rule. Occasionally the fever is high and continuous and the disease pursues a very rapid course not unlike that seen in cases of tuberculous broncho-pneumonia. A very high leukocyte count may be present, a point of some importance as a low leukocyte count or even a leukopenia is the rule in tuberculosis.

The disease is almost invariably fatal. But three cases have been known to recover. If the process becomes localized in a joint, amputatio or resection may prevent dissemination.

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[1] DICKSON: *Arch. Int. Med.*, December, 1915.
[2] *Trans. Amer. Climat. and Clin. Assoc.*, 1915.
[3] *Jour. Am. Med. Assoc.*, 1907, xlix, 1071.

Physical Signs.—These differ in no respect from what one finds i cases in which the pulmonary infiltration is due to tubercles.

PULMONARY ASPERGILLOSIS

Etiology.—A rare form of mould infection is that known as aspergillosi the exciting cause, in most instances, being the *aspergillus fumigatu* A monograph by Rénon[1] is the most authoritative study yet made this disease. Most of the reported cases, both in man and in animal have occurred in France. The occupation of the individual is of son importance as it has been observed chiefly in those who handle grai flour or meal. Men engaged in the forcible feeding (mouth to beal of pigeons, and hair-combers, who use flour to remove the grease from tl hair, have also acquired the disease.

The disease is extremely rare in this country. Osler has reported case in which the mycelium and spores of the aspergillus were presen in the sputum at intervals for a period of twelve years. The patien was in good health having no symptoms other than cough and sligh expectoration. In Holden's[2] case there was involvement of the lung and the cervical and axillary lymph nodes. In this case the organism was identified as the *aspergillus nidulans.*

Morbid Anatomy.—The apices of the lung are apt to be most involved The pulmonary lesions occur in the form of nodules, from 2 to 4 cm. ir diameter, which project from the cut surface. Emphysema may b present about the nodules. In the latent type of the disease considerabl fibrosis occurs with dilatation of the bronchi. The walls of the dilate bronchi contain small white or yellowish-white bodies which consist o mycelial threads. These bodies may appear in the sputum.

Aspergillosis may occur also in the lymph nodes and as a secondar infection in chronic bronchitis, bronchiectasis or pulmonary tuberculosis Under these circumstances its presence would not be suspected unles the sputum were especially examined.

Symptoms.—There is nothing distinctive in the symptomatology c the disease. The onset is as a rule gradual with malaise, loss of strengt and weight, anorexia, a slight evening rise in the temperature and pleuriti pains. The cough is at first dry and unproductive, but is soon attende with a frothy expectoration which later becomes greenish and purulen In two cases observed by Emerson[3] the sputum contained blackish specks Cultural studies showed the presence of the *aspergillus fumigatu* The sputum is often blood-tinged and the first intimation of pulmonar mischief may be the occurrence of a frank hemoptysis. Arrest of th disease accompanied by fibrosis of the lung and disappearance of th aspergillus often occurs.

Physical Signs.—In the early stages the physical signs are those c bronchitis. Later there is evidence of localization, which may be at th apex or the base of one lung. The physical signs are then those of a infiltration, such as occurs in tuberculosis.

Diagnosis.—The disease is to be distinguished from tuberculosi or infections of a similar nature. This is possible only by examinatio of the sputum for organisms other than the tubercle bacillus.

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[1] "Etude sur l'aspergillose chez les animaux et chez l'homme," Paris, 1897.

[2] *Trans. Amer. Climat. and Clin. Assoc.*, 1915.

[3] *Trans. Assoc. Amer. Phys.*, 1918.

PULMONARY SPOROTRICHOSIS

Sporotrichosis is a mycotic affection characterized by cutaneous and ternal lesions. It is caused by several species of parasitic fungi belong- g to the sporotrichosis group. The first case was described by Schenck, America, in 1898. Five years later de Beurmann and Ramond re- rted the first case in France. The most complete study of the affection to be found in the monograph by de Beurmann and Gougerot.[1] From is source most of the following facts have been obtained. Infection of e lungs with one of the Sporotrichosis group is exceedingly rare. hulmann and Massow[2] state that there are now five cases on record. three the organism was found in the sputum and in two it was re- vered from fluid obtained from the lung.

Etiology.—The most common site of the infection is the skin, espe- ally that of the hands and forearms. More rarely it occurs on a mucous embrane. Although healthy persons may be attacked it occurs most equently in individuals whose resistance has been lowered by some ronic disease. In a number of cases an abrasion in the skin has been resent which probably serves as the point of entrance. As the sporo- ichum is found on various vegetables, infection is most apt to occur in ose handling these food stuffs. One of the pulmonary cases ascribed r trouble to the inhalation of quantities of dust in the coffee mill in hich she was employed.

In 1912 Hamburger[3] reported 58 cases; two years later Sutton[4] added more from the literature and 5 personal observations. Of the 73 ses, none of which involved the lungs, which have been observed in this untry, 68 came from the region comprising the Mississippi River basin. hether this restriction of the disease to a certain area is due to local nditions or a failure to recognize it in other localities, is not clear. he same thing has been noted in regard to coccidoidal granuloma and astomycosis.

Morbid Anatomy.—Little is known as to the pulmonary changes. s the disease is chronic and may last for a year or more it is probable at fibroid-changes in the lung are a marked feature. Experimentally de eurmann and Gougerot were able to produce a variety of pulmonary sions in animals. These consisted of broncho-pneumonic areas, small odules, fibroid changes, abscess formation and in some instances cavity rmation similar to that seen in tuberculosis.

Dissemination from the skin lesion to the internal organs occasionally ccurs. The skin lesion, as described by de Beurmann and Gougerot, nay be one of three types: (1) It may appear in the form of multiple ummata in the subcutaneous tissues in various parts of the body. The gummata may be localized in one region of the body, especially the imbs, or be widely disseminated. The gumma begins as a small, hard, novable nodule which softens, gradually breaks down, and discharges viscid pus. (2) The lesion may be ulcerative and bear a strong re- emblance to cutaneous tuberculosis. There may be a small localized roup of ulcers. The usual location is on the hands and arms. (3) There may be a hard chancroid-like body, eroded on the surface.

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[1] "Les Sporotrichoses," 1912.

[2] *Bul. de la Société Médicale des Hôpitaux*, July 19, 1918.

[3] *Jour. Am. Med. Assoc.*, Nov. 2, 1912.

[4] *Ibid.*, Oct. 3, 1914.

Dissemination occurs through the lymphatics.

Symptoms.—The symptoms of the proven and suspected pulmonary cases are those of a chronic pulmonary infection. In the case reported by [illegible] and [illegible], a woman, aged forty, had been suffering from pulmonary symptoms for several months. She had a painful cough, mucilage-like sputum, which later became yellowish in color and contained small white granules. She also suffered from progressive shortness of breath and pain in the right chest. Physical examination showed impairment of the percussion note, rough breath sounds and râles at the right apex. The sputum was persistently negative for tubercle bacilli. Cultural studies of the sputum showed a sporotrichum closely resembling that described by de Beurmann. Agglutination tests confirmed this finding. The woman had been employed in a coffee mill. She associated her trouble with the inhalation of large quantities of dust in her place of employment.

Diagnosis.—This is possible only through bacteriological studies. In regions in which the infection is known to exist it should be borne in mind as a possible cause of an obscure pulmonary lesion.

ACUTE LOBAR PNEUMONIA

(Croupous Pneumonia, Fibrinous Pneumonia, Pleuro-pneumonia, Lung Fever)

The term *lobar* is used when the process involves an entire lobe or part of a lobe without healthy tissue intervening, in contradistinction to *lobular* pneumonia which occurs in isolated or confluent patches. The terms fibrinous or croupous, which are often employed, indicate the nature of the inflammatory exudate. The name pleuro-pneumonia is sometimes used to indicate that the pneumonic process is accompanied by a pleurisy. By the laity the disease is often referred to as lung fever or inflammation of the lungs.

Etiology.—Considered from both the morbidity and the mortality rates croupous pneumonia is decidedly one of the most important of the acute infectious diseases. As a cause of death it ranks with tuberculosis of the lungs, cardio-vascular disease and nephritis.

Age.—The disease may occur at any age period. Of 32,081 cases collected from the literature by Norris 40.2 per cent. occurred between the tenth and thirtieth years. Up to the sixth year the morbidity rate is quite high while the mortality rate is relatively low. Among those of advanced years the incidence is very high and the death rate equally so.

Sex.—In adult life males are more subject to pneumonia than females. Of 12,008 cases collected by Musser and Norris, 73 per cent. were males and 26 per cent. females. Among young children the two sexes are about equally effected. The greater incidence among adult males is probably due to the nature of their occupations which in many instances, offers greater chances for exposure.

Race.—In the United States it has long been recognized
monia is far more fatal among the negroes than the whites.
been noted also among the negroes employed in the construc
Panama Canal and among the negroes working in the mines of
South Africa. In both these places it has been noted that the
from pneumonia is highest among the recent arrivals and the
rate rapidly diminishes after a short residence in the community

Seasonal Incidence.—For years the relationship between cold and pneumonia has been regarded as a close one. It is well known that the disease very frequently follows, sometimes within a few hours, a wetting or a chilling as the result of some unusual exposure. The pneumococcus s a frequent inhabitant of the upper respiratory tract in a very large proportion of healthy people, different observers obtaining from 80 to 0 per cent. positive results. Ordinarily no trouble occurs but if the ndividual who harbors the organism happens to become chilled the resistance of the bronchial and pulmonary tissues may be sufficiently owered to permit the pneumococcus to gain a foothold. In other words, cold is a factor and not the actual cause of the disease. Climate does not seem to exert any great influence, the disease occurring with equal frequency in both hot and cold countries. Statistics, however, show that the incidence of the disease in the temperate zone is unquestionably higher during the winter and early spring months. This is to be ascribed to the cold in the winter months and to the wide variations in temperature which often occur in the early spring. In Philadelphia and New York and the Atlantic Coast generally, the highest percentage of cases occurs in February and March.

Influence of Insanitary Conditions.—The disease is more common in cities than in the country. There are several apparent reasons for this. City dwellers are confined to the house more closely than those in the rural districts and during the winter months the urban house is habitually overheated thus favoring the occurrence of catarrhal processes in the air passages.

Bad ventilation, dark rooms and overcrowding which characterize the housing conditions of a large portion of our city slums are undoubtedly factors in causing pneumonia. The influence of overcrowding is well shown by Gorgas[1] from his experience at Panama in 1906 and 1907 and also a few years later among the miners of the Rand, South Africa. Prior to 1907 the negroes working on the Isthmian Canal were housed in barracks. In 1907 the negroes were removed from the barracks and allowed to scatter along the line of the Canal, each man building his own hut for himself and family. Coincidently with the change in the method of housing the negroes, there was an immediate drop in the death rate rom pneumonia. In 1906 the mortality from pneumonia was 18.74 per thousand, in 1907 it was 10.65 per thousand and in the years succeeding up to and including 1912 the rate did not exceed 2.60 per thousand. This Gorgas attributes to the change in housing conditions. His investigation of the Rand miners also seemed to indicate that the crowded ving conditions under which these men lived had an undoubted influence n the high morbidity and mortality rate of pneumonia. Anders, in is study of the distribution of pneumonia in Philadelphia, found that he highest mortality from the disease occurred in the most densely opulated wards.

Epidemics.—There is now sufficient evidence at hand to indicate that neumonia may, in some instances, be transferred from person to person the contact is a close one. This probably accounts for the epidemics hich have been noted from time to time in overcrowded barracks, jails d almshouses. In the vast majority of instances pneumonia is to be garded as an endemic rather than an epidemic disease and for the most part cases occur sporadically. Occasionally, however, the disease attacks, within a short time, a number of people in a neighborhood or institution and has every appearance of being epidemic in character. Small localized epidemics have been noted also in institutions and for this reason many hospitals isolate their pneumonia patients.

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[1] *Am. Jour. Med. Sc.*, 1914, lxii.

The belief that pneumonia is contagious is strengthened by the observations of Dochez and Avery.[1] Although, as previously stated, pneumococci exist in the mouth of many healthy people such organisms can be distinguished from the more virulent ones which give rise to severe forms of lobar pneumonia. Dochez and Avery found that healthy persons intimately associated with cases of lobar pneumonia harbored the disease-producing types of pneumococci and that in every instance the organism isolated corresponded in type with that of the infected individual. Convalescents may carry virulent pneumococci for some time.

Personal Conditions.—While robust healthy men are frequently attacked by pneumonia, individuals who have become debilitated through disease or dissipation are far more susceptible to the disease. A factor which may predispose to pneumonia is acute fatigue due to violent exercise such as rowing or running in competition as Cowles[2] has pointed out. He suggests that this might have been a contributing cause to pneumonia in the army camps and that the disease might be lessened by preventing fatigue in persons not in good physical condition. Among those suffering from the various chronic diseases such as arterio-sclerosis, nephritis, heart disease, malignant disease, etc., an attack of croupous pneumonia is the terminal event in a very large number of cases. The extreme susceptibility of the alcoholic to a fatal attack of pneumonia has long been recognized.

Trauma.—Pneumonia has been noted in a small percentage of cases to follow some injury of the chest wall without there being any injury of the lung itself.

Previous Attacks.—Lobar pneumonia is common with diphtheria and erysipelas, differs from the other infectious diseases in that one attack does not afford protection against a recurrence of the trouble. No other disease is so prone to recur in the same individual. Two and three attacks have been noted frequently and not a few instances are on record of individuals who have had ten or more attacks. In the oft-quoted case of Benjamin Rush no less than 28 attacks occurred.

Post-operative Pneumonia.—It is not altogether clear whether the combined effect of an anesthetic, particularly ether, and operative interference are factors in the production of lobar pneumonia. In a smal percentage of cases pneumonia undoubtedly does follow the operation Laparotomies seem to be followed by pneumonia more frequently than is the case when other portions of the body are operated upon. It is to be borne in mind, however, that a latent and unrecognized pneumonia may have been present at the time of the operation.

The generally accepted theory as to the cause of post-operative pneumonia is that one or all of the following factors may be at fault: (1 The patient may become chilled on the operating table; (2) that excretions from the mouth may become aspirated into the lung; and (3) that the infection may arise from an unsterilized inhaler.

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[1] *Jour. Exp. Med.*, xxii, 1915.

[2] *Boston Med. and Surg. Jour.*, Oct. 31, 1918.

Inhalation and Aspiration Pneumonia.—Lobar pneumonia may arise as the result of the aspiration of fluids or solid material into the lung or as the result of the inhalation of irritant dusts or chemicals. But as a rule the above-mentioned accidents are followed by broncho-pneumonia rather than the lobar type of the disease.

Occupation.—This is often given as a predisposing cause of pneumonia. The effect of any particular occupation on the production of pneumonia is problematical. If the sanitary conditions, which include proper ventilation, are good the occurrence of pneumonia among the workers will be less than if the reverse be true. It is also to be borne in mind that the sanitary arrangements of the working place may be perfect while the home conditions are very bad.

Bacteriology.—The exciting cause of lobar pneumonia, in a large proportion of cases, is the pneumococcus. This organism is also known as the diplococcus pneumoniæ and micrococcus lanceolatus. In a few instances the pneumonia is caused by a streptococcus or the influenza bacillus; more rarely Friedlander's bacillus is the organism found. These organisms may occur alone or in association with the pneumococci.

For many years it has been a matter of clinical observation that epidemics of pneumonia showed marked differences in the mortality rate and that even in the same epidemic the virulence of the infection varied greatly. To this variability of the disease may be ascribed some of the favorable reports which have appeared from time to time on the effect of some particular method of treatment.

As the result of work by Neufeld and by Cole and his co-workers, Dochez, Avery, and Gillespie, at the Rockefeller Hospital, it has been shown that there are several varieties of pneumococci and that these can be distinguished one from the other by immunological reactions. Thus by testing pneumococci with the serum of animals immunized against them, it has been found that they may be divided into at least four groups. According to Cole[1] all the organisms of group I are alike so far as their immune reactions are concerned. The same is true of organisms of group II. In group III (pneumococcus mucosus) the organisms also show peculiar immunity reactions, and in addition, they differ from those of the preceding groups in that they have larger capsules and form a sticky exudate in animals. In group IV are placed the remaining organisms which are found to show no common immunity reactions and are, in addition, much less virulent.

Although the clinical features show no marked differences, the severity of the pneumonia varies greatly with the type of organism. The following figures given by Cole[2] show the relative frequency and the mortality of pneumonia, due to the different types of pneumococci, observed n the Rockefeller Hospital, New York.

Etiological agent	No. of cases	Percentage incidence	Mortality
neumococcus type I	78	35.0	25.0
neumococcus type II	75	33.5	29.0
neumococcus type III	22	9.0	45.0
neumococcus type IV	48	21.5	12.5
Total	223		

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[1] *Monthly Bulletin N. Y. Dept. of Health*, February, 1916.
[2] *Trans. Assoc. Am. Phys.*, 1915, p. 234.

In a study of 195 cases at the Pennsylvania Hospital, Philadelphi Richardson[1] obtained the following results:

Etiological agent	No. of cases	Percentage incidence	Mortality
Pneumococcus type I	60	31	30
Pneumococcus type II	39	20	25
Pneumococcus type III	13	6	50
Pneumococcus type IV	83	43	12
Total	195		

It will be seen that the figures in the two series are very similar a: that the majority of the cases are due to types I and II, and that t mortality ranges from 25 to 30 per cent. Infection with type III is t least frequent, a fortunate circumstance, as the mortality in this gro is very high. As Cole points out the frequency of cases due to type probably varies in different places and at different times. The mortali in group IV is not high; in some series it has been as low as 6 per cent.

The separation of pneumococci into distinct groups has been of gr practical value.

1. A knowledge of the type of organism has made it possible to fo tell the probable outcome of the disease. Thus if the agglutination t shows the presence of type I or II the use of a specific serum is possib if type III is present the outlook is bad; if the organism belongs to ty IV the chances are greatly in favor of the patient. To determine t type a mouse is injected with some of the sputum and 6 hours lat some of the peritoneal fluid is removed and an agglutination test is don This may determine the type at once. If, however, no reaction occu the mouse is allowed to die, this taking place within 24 hours, and t agglutination test is again done. Cultural peculiarities may be an a also. It is needless to say that this is possible only in a hospital with well-equipped laboratory.

2. It has been known for some years that pneumococci are prese in the mouths of healthy individuals in a very high percentage of case some observers have found as high as 80 or 90 per cent. of normal ind viduals harboring these organisms in the mouth. This has made difficult to understand how these bacteria can cause pneumonia unle there is some other primary or predisposing factor. Cole offers t following explanation: For a pulmonary pneumococcus infection occur, at least two factors are probably responsible. First, the conditi in the host, local, general or both, must be favorable for infection; a second, an organism of suitable virulence must be present. With orga isms of the type ordinarily found in the mouth (type IV), probably t changes in the patient are of chief importance. With pneumococci of t fixed types (I, II, and III), however, the presence of the organism is great importance, and may even determine the onset of the illne Even in this case, however, changes in the resistance of the patients probably of some importance, as, under certain circumstances, healt persons may carry these fixed types without becoming themselves fected. This last-mentioned group may be compared to the so-cal diphtheria and typhoid carriers.

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[1] *Trans. Assoc. Am. Phys.*, 1915, p. 234.

3. The most important outgrowth of this work is that which relates to treatment. It is too early to speak with certainty but the results so far obtained with the serum produced at the Rockefeller Hospital are extremely encouraging. Cole states that the most successful results have been obtained in cases treated with serum of type I. Among the cases so treated the mortality was only 8 per cent., as contrasted with 25 per cent. in the untreated cases. The results obtained in a few cases treated with serum of type II have been much less encouraging. There is no specific treatment for either type III or type IV.

Morbid Anatomy.—As the result of careful bacteriological studies it is now apparent that croupous pneumonia is a generalized infection with a local manifestation. And while the brunt of the infection is borne by the lung the diplococcus may be found in various other portions of the body as well as in the circulating blood. We have already alluded to the fact that a very high percentage of healthy individuals harbor the diplococcus in the upper air passages and that under ordinary conditions the germ is innocuous.

If, however, the individual becomes chilled or is the victim of some chronic disease the natural defenses are incapable of maintaining the upper hand. The result is that the diplococcus is unleashed, as it were, and permitted to grow unrestrained.

Since the time of Laennec it has been customary to describe the changes which take place in the lungs as: (1) the stage of engorgement; (2) the stage of red hepatization; and (3) the stage of gray hepatization. In accepting this classification, however, it is to be understood that the development of the different stages of the morbid process is not always uniform. And while for the most part the lesion may be predominantly that of engorgement or red or gray hepatization, two or all three of these stages may be present in the same lung.

In the stage of *engorgement* the affected portion of the lung is deep red in color, heavier than normal and often slightly edematous. Although the air content is diminished the tissue still crepitates and excised portions will float in water. On section there is an abundant hemorrhagic exudate. The capillaries are distended and the alveolar epithelium swollen and occasionally desquamated.

The second stage or that of *red hepatization* is characterized by coagulation of the fibrinous exudate and the affected area has become of the consistence and appearance of liver, hence the name hepatization. In this stage the lung is swollen and heavy; pits on pressure; is very friable and readily broken. It is completely airless and excised portions sink in water. On section the cut surface is dry and presents a granular appearance due to the projection of small fibrinous plugs in the air cells. A tenacious, creamy, grayish-red fluid containing granular masses can be scraped off the cut surface. Fibrinous casts are often found in the smaller bronchi. Microscopically the air cells are seen to be filled with a coagulated exudate in which there are swollen and desquamated epithelium, red blood cells and a few leukocytes. A round-celled infiltration is also seen about the vessels in the interlobular septa.

The transition from the stage of red to *gray hepatization* is gradual. The red color is replaced by a yellowish or ashen gray color. Here and there are often to be noted dark red or reddish gray areas and if in addition anthracosis is at all marked the cut surface presents a mottled appearance

(Fig. 272). The lung is swollen and firm to the touch and is often indented with the impressions of the ribs. The tissue is more friable and more easily torn than in the second stage.

The overlying pleura is dull and granular in appearance and covered with a fibrinous exudate. A small effusion may be present (see Fig. 334). Inflammatory changes in the pleura are constantly present in lobar pneumonia except in those cases in which the lesion is centrally located and does not approach the periphery of the lung.

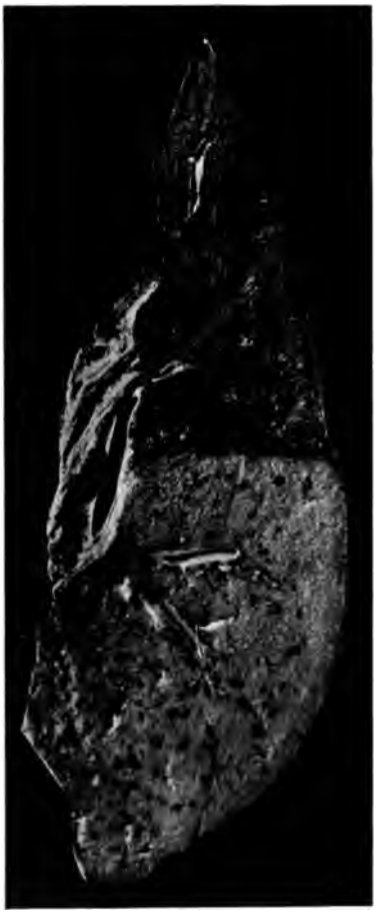

Fig. 272.—Gray hepatization with anthracosis.

On section the cut surface of the lung has become moister and the granular appearance less marked. The fluid scraped from the surface of the section is more abundant, milky and puriform. Microscopically the air cells are seen to be packed with leukocytes while the fibrin and the red blood cells have largely disappeared. In some instances the tissue is very soft and freely exudes a purulent exudate (purulent infiltration) and small abscesses may develop.

The anemic appearance of the affected area during the stage of gray hepatization is due in part to the large number of leukocytes and in part to the pressure on the capillary vessels.

While the majority of cases follow the sequence of events described above, each stage lasting from two to three days, the process may terminate in two or three days instead of being prolonged for a week or more. In children, the aged, and the asthenic, the amount of fibrin produced may be small and the alveoli but slightly distended, so that the usual dry, granular appearance of the lung is not observed (Adami).

Resolution.—The first evidence pointing to the stage of resolution is a breaking up of the fibrin and fatty degeneration of the leukocytes. The lung becomes smaller, has a boggy feel and the pleura becomes relaxed and thrown into folds. On section the surface is yellowish in color, very moist and a creamy, almost purulent fluid can be squeezed out. The softened exudate is removed partly by being expectorated and partly by absorption; the latter is probably the more important as the exudate disappears in cases in which both cough and expectoration are entirely absent. The dissolving of the exudate is due to autolysis.

The coagulated albumen is broken up into soluble albuminoids and further decomposition products through the action of a ferment that is probably given off by the leukocytes (Strümpell). Cook, in Osler's Clinic, has shown that the excretion of the dissolved exudate takes place through the kidneys as shown by the excessive nitrogen excretion in the urine.

With the removal of the exudate the desquamated epithelium in the alveoli are replaced by regeneration from those remaining intact; gradually there is a complete restoration to the normal. In the vast majority of cases, especially in young, healthy individuals, complete resolution takes place uninterruptedly. In a small proportion of cases the diseased area instead of undergoing a normal involution becomes the site of an abscess, a gangrenous process, or fibroid induration.

Site of the Disease.—It has been shown in every series of cases of lobar pneumonia that the lower lobes of the lungs are the parts most frequently affected and that the right lower lobe is slightly more vulnerable than the left. The excess of frequency of the affection of the right over the left lung as a unilateral affection, and independently of the location of the lesion, may be regarded as occurring in the proportion of about 3 to 2. The distribution of the lesions is well illustrated in Jüsgensen's[1] large series of 6666 cases: right lung, 3580 cases: left lung, 2548 cases; both lungs, 538 cases. Involvement of the upper lobes occurs more frequently in children and in those of advanced years than in the middle periods of life. When the disease attacks both lungs it is the two lower lobes that are usually affected but bilateral lesions may occur in both apices or in one apex and one lower lobe. Rarely both lungs may be extensively involved. In a case seen at the Philadelphia General Hospital death occurred on the third day and at the autopsy both lungs, from apex to base, were found to be in the stage of red hepatization.

The unaffected portion of the lobe may show no change but is very commonly congested and edematous. The uninvolved lobes both on the affected and the unaffected side may be dry and bloodless or may show evidences of congestion.

Associated Lesions.—The mucous membrane of the bronchi is more or less congested and the smaller tubes are usually blocked with a fibrinous exudate. In some instances even the large bronchi are filled with the exudate constituting what is known as massive pneumonia. The bronchial lymph nodes are constantly swollen.

Except in cases of central pneumonia the pleura is always involved. The portion overlying the pulmonary lesion is dull, and granular in appearance and covered with lymph. A slight excess of serofibrinous or fibrinopurulent exudate is often present and occasionally becomes sufficiently large to constitute a true empyema. The occurrence of the latter is relatively common in children.

Pericarditis.—Based on autopsy findings pericarditis is far more common than clinical data would indicate. During life pericarditis is almost constantly overlooked. This is well illustrated by figures given by Cowan who reports a post-mortem incidence of 12.6 per cent. as contrasted with 1.2 per cent. from the wards. The pericardium may be involved as a part of the general infection but in most instances the pericardial infection arises apparently as the result of direct extension from the lung. Rarely a pericardial effusion, serofibrinous or purulent in character, may occur.

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[1] Ziemssen's "Handbuch d. Spec. Path. u. Ner.," 1874.

Endocarditis is not often noted during life but at the autopsy table it is not an infrequent finding. In a series of 100 post-mortem cases Osler noted the presence of endocarditis in 16 instances. The lesion is more commonly of the ulcerative type.

Meningitis.—Occasionally meningitis occurs during the course of lobar pneumonia. The frequency with which it is encountered varies widely. Osler noted the presence of meningitis in 8 of 100 autopsied cases while Pearce observed but 2 out of 121 cases.

Of the more unusual complications mention should be made of peritonitis, otitis media, arthritis, parotitis, phlebitis and abscess formation in various portions of the body. In addition there may be associated with the disease diphtheritic inflammation of the mucous membrane of the stomach, intestines or mouth.

In common with the other infectious diseases degenerative changes are usually evident in the heart, liver and kidneys and the spleen is often enlarged and softened.

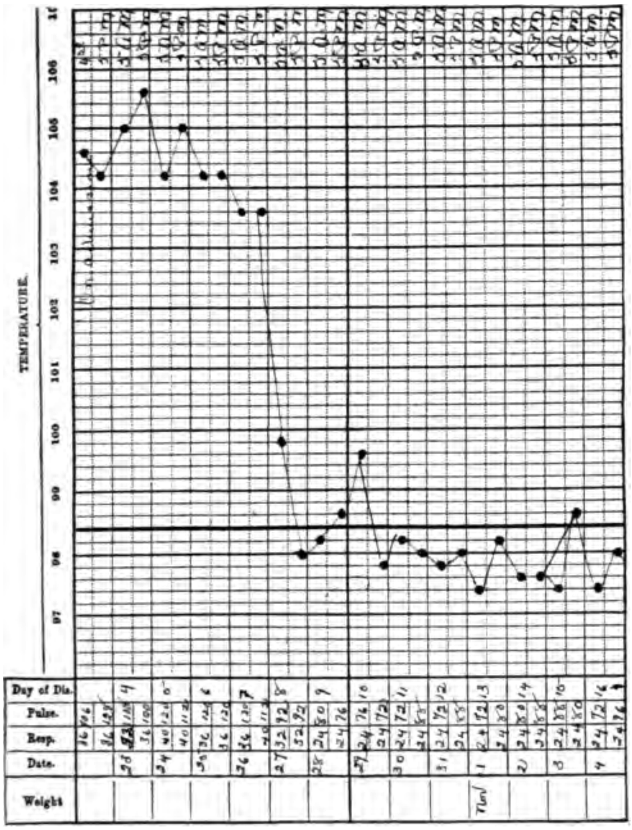
Symptoms.—The incubation period of pneumonia is not absolutely known. The disease has been observed to develop within a day or two after a traumatic injury to the chest. In those instances in which the disease assumes the proportions of an epidemic seven or eight days may elapse between the time of exposure and the development of the trouble.

In the great majority of cases pneumonia both in its mode of onset and in the symptoms which characterize its course, presents a typical picture. It is estimated that in 80 per cent. of cases the onset occurs suddenly. In 20 per cent. it is indefinite and the actual occurrence of the disease is preceded by prodromes. In many of these cases there is a history of a slight respiratory infection which differs in no way from an ordinary "cold." This may have been present for several days or even a week or longer when the pneumonic process suddenly manifests itself. In other instances the patient may complain for several days of a feeling of lassitude, headache, pain in the limbs, chilliness or slight fever. An insidious onset is frequently seen in pneumonia as it occurs in the aged, in alcoholics and as a terminal infection in chronic diseases.

In frank, typical, lobar pneumonia the onset is sudden and characterized by a severe chill which may last for as long as a half an hour; or instead of the initial rigor the patient may complain of chilliness and pain in the side. Coincidently with the chill, fever appears. By the end of the first day or the beginning of the second the clinical picture in a typical case is characteristic. The patient is breathing more rapidly than usual; complains of pain in the side, the pain in some instances being almost unbearable; and there is a frequent, partially suppressed cough which for a day or so is unproductive. By the second or third day the patient expectorates the characteristic sputum. This may be bright red but more commonly is brownish-red or rusty in appearance and extremely tenacious. The gummy, viscid character of the sputum, which renders its expulsion difficult and causes it to adhere to the side of the sputum cup, is pathognomic of croupous pneumonia.

The disease has an average duration of from five to ten days when, in a little over half of cases, it abruptly terminates by crisis.

Special Symptoms.—*Fever.*—A rise in the temperature occurs at time of the chill and may reach the fastigium within 12 to 24 hours. ome instances the fever may increase gradually for a couple of days. temperature of pneumonia is continuous in type with slight evening issions which may not amount to more than one degree. In the rage case the temperature ranges between 103° and 105°F. during the rse of the disease when it abruptly falls by crisis (Fig. 273). About er cent. of cases may be expected to terminate in from five to ten



273.—Crisis in lobar pneumonia. Note the drop in the pulse and respiratory rates following the fall in the temperature.

Occasionally cases are encountered in which the disease termi- s as early as the third day and more often it may be prolonged for weeks or more. In cases of long duration the temperature usually ides by lysis (Fig. 274). In not a few cases there is apt to occur in middle of the course of the disease a slight drop in the temperature r which it again arises. This is known as a pseudo-crisis (Fig. 275). ng 1854 collected cases a pseudo-crisis was noted in 172 or 9.8 per . (Norris). Occasionally just prior to the crisis the fever may increase tly.

While as a rule hyperpyrexia is associated with fatal cases recovery has followed very high temperatures—107° to 109°F. A fairly high degree of fever, from 103° to 105°F., is not a bad sign but relatively low temperatures are very commonly associated with fatal cases. A temperature of 100°F. or less is very frequently seen in pneumonia as it occurs in the aged, in the debilitated and in those with an overwhelming infection.

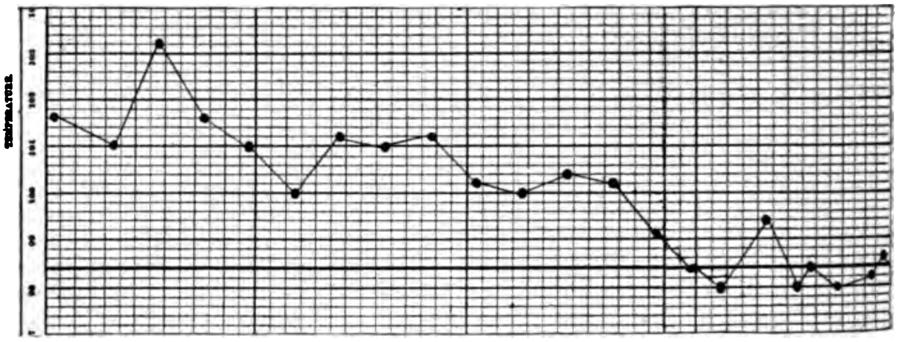


Fig. 274.—Fall of temperature by lysis.

Pain in the chest is one of the most constant symptoms of pneumonia, some observers having noted its presence in as high as 90 per cent. of cases. It may be the first symptom noticed, in some cases preceding the chill; on the other hand, it may not make itself manifest until the third or fourth day of the disease. The pain may disappear in a day or so but as a rule continues throughout the attack. In the frank pneumonia of adults the pain is usually sharp and lancinating in character and in some instances is so severe as to be almost unbearable. In central and apical pneumonias and in children pain may be very slight or entirely

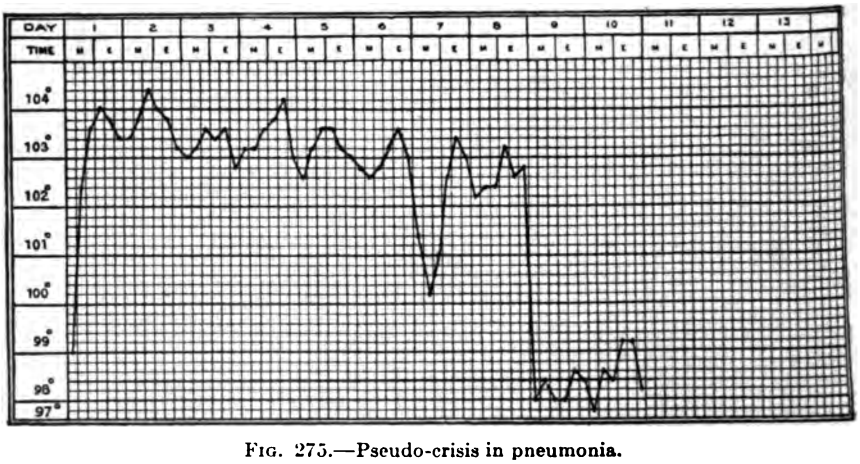


Fig. 275.—Pseudo-crisis in pneumonia.

absent. Nor is it much in evidence in senile or asthenic pneumonias. In delirious patients pain is not complained of at all. As a rule the pain corresponds to the site of the consolidation but not infrequently it is referred to the region of the nipple on the affected side. Rarely pain may be complained of on the unaffected side. Most important to bear in mind is the fact that the pain produced by pneumonia may be referred to the abdomen and thus simulate appendicitis or some other abdominal lesion. Failure to recognize the true source of the pain has, in not a few instances, led to the performance of a laparotomy. Chatard noted abdominal pain 51 times in 658 pneumonia patients (7.7 per cent.) and Griffith has recorded a number of instances in which it has occurred in children.

While severe pain in the chest is not pathognomonic of pneumonia its occurrence is significant if the pain is greatly increased by breathing, coughing and talking, or if aggravated by moving about. In some instances the chest wall over the site of the consolidation is hyperesthetic, the patient often complaining bitterly when the region is percussed. Immobilization of the affected side often relieves the pain by limiting the respiratory excursion and thus preventing the inflamed surfaces of the pleura from rubbing together.

Headache is usually experienced at the onset of the disease. In some instances it is very severe. Earache may also be complained of.

Respiratory Symptoms.—Acceleration of the respiratory rate may be one of the earliest symptoms of pneumonia especially when the disease occurs as a terminal infection in the aged or in those suffering from some chronic affection. The type of the breathing is of the greatest diagnostic importance. The respirations are short and shallow and often accompanied by an "expiratory grunt" and inspiratory dilatation of the alæ nasi. The respiratory rate is almost invariably quickened. It is rarely under 30 and in adults the rate may be as high as 60 or 70 per minute. In children the respiratory rate may be 100 or more. Quickening of the respiratory rate is due to several causes, namely: to a limitation of the respiratory excursion due to pain, which is especially noticeable when one of the lower lobes is involved; to the amount of pulmonary tissue consolidated; and to fever, toxemia or cyanosis. In those cases in which pain is the chief factor the respirations tend to become deeper and slower with the subsidence of the pain; on the other hand, if the involvement extends or if the toxemia increases in severity the respiratory rate will increase in rapidity. While extreme rapidity of the respiratory rate cannot be regarded in all cases as of serious significance it is to be noted that a fatal issue occurs more frequently in patients in whom the rate is above 50. The exceptions to this general rule are cases in which the rapid and shallow breathing is caused by severe pain. Dyspnea may be present in conjunction with the increased respiratory rate. It is usually indicative of a profuse bronchial catarrh, edema of the lungs or some complication which restricts the expansion of the lung, such as an effusion.

Cough.—In the atypical forms of pneumonia, such as sometimes occur in the aged, in alcoholics and in those with apical lesions, cough is sometimes absent, but in practically all cases of frank pneumonia in adults cough is a prominent symptom. In those cases in which the pneumonia is preceded by a "cold" the cough is already present at the time of the chill. When the disease is ushered in with a sudden onset the cough occurs with or very shortly after the chill and in the beginning is unproductive and often paroxysmal. Owing to the associated pleurisy the act of coughing greatly intensifies the pain and for this reason the patient attempts to suppress or smother the cough. In the course of one to three days expectoration appears and the cough may lessen in severity. On the other hand, the cough may increase in severity and greatly tax the patient's strength. The sudden disappearance of the cough is to be looked on as of grave import as it not infrequently indicates the onset of exhaustion or coma.

Sputum.—The character of the sputum constitutes the one feature of pneumonia that may be regarded as pathognomonic. Often absent for the first day or so, it appears first as a frothy, white substance, which may be streaked with blood. In the course of a few hours, however, it assumes its characteristic appearance. In color it is rusty or brick-dust in appearance, due to the admixture of blood and the fibrinous exudate. Of more importance is the consistency. Typical pneumonic sputum is semitransparent, viscid and extremely tenacious, being expelled with difficulty and adhering to the side of the sputum cup. As the disease progresses and the stage of gray hepatization is reached, the sputum loses its sanguinolent appearance and becomes yellowish and finally white. At times it has a greenish tinge. Instead of being thick and tenacious, the sputum may be watery and of a brownish color, resembling prune juice. Sputum of this character is usually associated with cases of severe infection such as are encountered among habitual drunkards.

Rarely the sputum shows no evidence of blood throughout the attack and still more rarely the sputum may be absent. The latter is apt to be the case in children who swallow the sputum and in delirious or stuporous patients who fail to cough it up. While the expectoration of pure blood may occur in pneumonia, the occurrence of a frank hemoptysis should arouse one's suspicions as to the possibility of a tuberculous rather than a pneumococcus infection.

Microscopically the sputum shows in the early stages the presence of crenated red blood cells; later leukocytes, shreds of fibrin and at times Curschmann spirals and casts from the smaller bronchioles. The type of organism has been dealt with under the section on bacteriology.

Nervous Symptoms.—As an initial symptom headache is common. In children the attack is not infrequently ushered in with a convulsion. During the course of the disease delirium is apt to occur. The character of the delirium varies greatly; in some instances it consists of slight incoherence; in others, especially in asthenic and senile patients, it is commonly of the stuporous and muttering variety; in still others the delirium takes the form of violent maniacal excitement often with homicidal tendencies. As the last-mentioned condition may develop suddenly pneumonia patients should always be carefully watched.

Among those addicted to the excessive use of alcohol the occurrence of delirium resembling delirium tremens is not uncommon. Apex pneumonias are said to develop a severe form of delirium more often than those with a lesion at the base of the lung. Among 7624 cases of pneumonia delirium was noted in 1343 or 17 per cent. (Musser and Norris).

The Blood.—There is no anemia which can be ascribed to the pneumonia and the red cells and hemoglobin show little or no change. There is, however, in the great majority of cases a leukocytosis. In the average case the white cell count ranges between 15,000 and 30,000; it may be as high as 100,000. Very high counts are often associated with cases of severe infection and are apt to terminate fatally. The absence of leukocytosis is a bad sign and often points to an unusually severe nfection or lack of resistance.

The Urine.—In practically all cases the quantity of urine is greatly reduced, highly colored, increased in specific gravity and markedly acid—he so-called febrile urine. Traces of albumen are usually present and here may be tube casts. The chlorides are greatly diminished or entirely absent.

Digestive Symptoms.—In children the onset may be attended with vomiting. The tongue is coated with a white fur and in toxic cases it may become very dry. The appetite is poor and the bowels usually constipated. Diarrhea may arise if the mucous membrane of the bowel becomes the seat of a croupous exudate. A very troublesome and often dangerous symptom is meteorism.

The Skin.—Labial herpes occur so frequently in croupous pneumonia that their occurrence is always of diagnostic value. The vesicles may appear at the angles of the mouth and about the chin or nose. Occasionally they occur about the genitals and anus. The frequency of herpes varies greatly, the figures ranging from 5 to 50 per cent. Cases in which herpes occur are said to have a better prognosis.

Sweating may occur during the course of the disease but as a rule is noted only at the time of the crisis when it may be profuse. Flushing of the cheek on the affected side is a common phenomenon.

The Crisis.—The phenomena which characterize the crisis or termination of an attack of lobar pneumonia constitute the most remarkable feature of this or any other disease. In the course of a few hours a patient who has been desperately ill and whose fate hangs in the balance wakes from a sound sleep nearly free from his distressing symptoms; and not less remarkable is the fact that with the extraordinary change in the subjective symptoms the physical signs over the affected lung remain unaltered. The cause of this sudden change is believed to be due to the establishment of an active immunity to the toxin of the pneumococcus.

As already stated the crisis may be expected on or about the seventh day of the disease; in some instances it occurs a day or so earlier and in others it is delayed for several days longer.

The onset of the crisis may be heralded by a disappearance of the restlessness and delirium; the flushed face becomes pale; and the anxious facial expression is replaced by one of calm. At the same time the patient becomes drowsy and falls asleep. The sleep at first broken gradually deepens and may last from 5 to 12 hours or more. During this time the temperature rapidly falls from 104° or 105°F. to 97° or 98°F. and is accompanied by sweating which is often most profuse. In some instances the temperature may fall six or eight degrees within 1 hour, while in others the decline lasts as long as 24 hours. The temperature may show a slight rise on the following day and then drop permanently. The respirations become slower, the pulse rate drops and the cough becomes ooser (see Fig. 273). The rusty character of the sputum may persist for a few days. If delirium has been a marked feature it may be several days before the mind is entirely clear. In a case free from complications the convalescence is rapid, recovery being usually completed in about three weeks.

The phenomena just described occur in about half the cases terminating favorably. In the remainder the fever instead of suddenly disappearing gradually subsides by lysis and at the same time there is a gradual rather than a rapid abatement of the symptoms. The more prolonged the attack the more apt is the termination to be by lysis.

Physical Signs.—The following description is that of frank lobar pneumonia as it occurs in the adult. The commoner variations which characterize the atypical case will be considered separately. The physical signs indicative of the disease manifest themselves in from 24 to 48 hours. If delayed beyond this period, it is usually due to the fact that the pneumonia starts in the central portion of the lung and gradually extends toward the periphery. In some instances the lesion does not extend beyond the central portion of the lung and physical signs may be absent throughout the entire course of the disease.

Inspection.—There are certain striking features about pneumonia even from its onset, that render the probability of its presence apparent from inspection alone. The breathing is rapid and the respirations are short and shallow, often accompanied by an "expiratory grunt," inspiratory dilatation of the alæ nasi and an exaggerated action of the accessory muscles of respiration, such as the sternocleidomastoids and scaleni. The presence of herpes labialis is very suggestive. One cheek may be more flushed than its fellow and very often the pupils are unequal, the dilated pupil being, as a rule, on the affected side.

The patient usually lies flat on his back but may lie on the affected or unaffected side. In the effort to minimize the pain the patient may be propped up in bed and inclined toward the affected side.

Inspection of the chest itself, especially when the disease is fully developed, will show a diminution of expansion on the affected side and an exaggerated movement on the opposite side. When the lower lobe is affected the expansion of the chest over the upper lobe on the affected side may be exaggerated. Occasionally when the left lung is the seat of disease the involved portion may pulsate synchronously with the heart. Inspection of the cardiac area shows nothing abnormal.

Palpation.—Diminished expansion of one side as compared to the other may be more apparent from palpation than inspection. During the stage of engorgement which usually lasts from 12 to 24 hours the tactile fremitus may show no change but during the period of consolidation it is exaggerated. Absent or diminished expansion occurs in cases of central pneumonia and in the so-called massive pneumonia when the bronchi are plugged with the fibrinous exudate. Occasionally a friction or rhonchial fremitus may be felt. Pressure over the consolidated area will often elicit a tender spot, the pain being referred to a point some distance away.

The *pulse* in sthenic lobar pneumonia is full and bounding once the disease has become established. During the chill it is small and in the aged or debilitated it may be weak and rapid throughout.

Formerly considerable emphasis was placed on the pulse-respiration ratio. In health this is about 4.5 to 1, while in pneumonia it may be 3 to 1, 2 to 1, or even 1 to 1. In an obscure case this ratio is suggestive of pneumonia as there is no other febrile disease in which the respirations are so frequently increased out of proportion to the pulse.

In the case of average severity the pulse ranges from 100 to 110. Generally speaking a low pulse rate is a favorable indication. When the rate is 120 or over the outlook is serious, as it has long been noted that a high pulse rate is attended by a very high mortality. The pulse rate in women tends to be slightly higher than in men. A high pulse rate in children does not have the same significance as in adults as the former not uncommonly have a rate ranging from 120 to 150. Irregularity of the pulse is also to be viewed as a very serious symptom. Of 17 patients with an irregular pulse 13 died (Lord).

A few years ago G. A. Gibson pointed out that when the *blood-pressure* as expressed in millimeters of mercury does not fall below the pulse rate as expressed in beats per minute, the patient is in no immediate danger. With, for example, a pulse rate of 100 and a blood-pressure of 125 there is no need for interference but if this ratio becomes reversed active stimulation is demanded in order to relieve the threatened cardiac failure. While this observation is of importance it is not to be depended on absolutely.

Percussion.—The percussion note over the affected lobe may, if the case is seen early, be tympanitic in quality. As the consolidation increases the note becomes absolutely dull and there is in addition, a feeling of resistance. The dull note remains even after the crisis but with the onset of resolution the note gradually becomes more and more resonant. Occasionally the note may become resonant within two or three days but as a rule several weeks elapse before the normal standard has been regained. Very often in percussing over the affected area varying degrees of hypersensitiveness are encountered due to the associated pleurisy. If the lesion is situated some distance from the periphery of the lung the note will show little if any impairment and may even be hyperresonant. Over the unaffected portions the note is resonant or if the consolidation is extensive the note may be hyperresonant as the result of compensatory emphysema.

Auscultation.—At the onset of the disease the breath sounds over the affected area may be slightly suppressed or even harsher than normal, and broncho-vesicular in character; usually they are somewhat suppressed. *The most important diagnostic sign during the first stage is the crepitant râle.*

The distinctive features of crepitant râles are that they produce a fine, crackling, high-pitched, sound, which occurs abruptly at the end of inspiration, especially a forced inspiration; they are never heard during expiration. The resemblance between the sound produced by a shower of crepitant râles and that by rubbing the hair between the fingers close to the ear is so similar as to make the comparison a very apt one. The crepitant râle is heard during the first day or so only.

The crepitant râle while a very important diagnostic sign in lobar pneumonia is not absolutely pathognomic as it may occur in a localized area at the apex as an accessory sign of phthisis. It occurs transiently under the following circumstances: A patient who has been confined for
time in bed, lying on the back and much enfeebled with any dis-
if suddenly raised to a sitting posture and auscultated, a crepitant
s often found on the posterior aspect of the chest at the end of forced

inspiration. The râle disappears after a few forced inspirations and is heard, not on one side only, but on both sides. It should be mentioned also that if the stethoscope be applied to a hairy chest the movements in the act of inspiration may produce a sound identical with the crepitant râle (Flint).

When the vesicles become entirely filled with the exudate and the lung presents a solid appearance no extraneous sounds are produced. When this stage is reached the breath sounds become tubular or bronchial in character, the inspiration being high-pitched and of the same duration as expiration. In the ordinary case of frank pneumonia the bronchial breathing is usually quite loud. The voice sounds are also intensified and exaggeration of the spoken voice and bronchophony are usually marked. The whispered voice is also distinctly transmitted. Both the spoken and whispered voice may have, at times, the peculiar quality known as ægophony. This sign is common in cases of pleural effusion but it may also occur over consolidated lung tissue. In cases of massive pneumonia both the breath and voice sounds may be very distant or entirely absent. Over the unaffected portions of the lungs the breath sounds are clear and slightly exaggerated but in those cases in which an attack of bronchitis has preceded the pneumonia râles may be present.

In examining the chest of an individual suspected of having pneumonia care should be taken not to overlook the axillary region. Very often when the pneumonia affects the upper lobe physical signs will be obtained only high up in the axilla.

With the onset of gray hepatization râles are again heard. The returning râle or *crepitus redux*, is held by some to be identical with the crepitant râle of the first stage, but others consider it coarser and lacking the distinctive features of the latter. In addition to the crepitus redux there are to be heard numerous subcrepitant râles. As the vesicles are emptied of their contents the character of the breath sounds gradually change; the bronchial breathing becomes broncho-vesicular in character and finally pure vesicular, although for a time the vesicular murmur is apt to be less intense. The restoration of the breath sounds to the normal may occur rapidly but, as a rule, weeks elapse before this is attained.

Careful examination of the heart should be a routine procedure in cases of lobar pneumonia. Early in the disease the heart sounds usually show no change unless toxic symptoms are marked from the onset. In the later stages of the attack functional murmurs are very common. In a few cases the murmur will be due to an acute endocarditis and, as has been shown in the section on morbid anatomy, this is not uncommonly ulcerative in character. The second pulmonic sound is generally accentuated and may be reduplicated as well. A diminution of the accentuation of the pulmonic second sound is to be regarded seriously as it is often a forerunner of acute dilatation of the right ventricle. Cardiac failure may manifest itself by sudden syncope but more often it develops more or less gradually. The pulse becomes more rapid and in addition there are cyanosis, dyspnea and cold extremities. The above symptoms may occur at or about the time of the crisis; occurring at this time they are, as a rule, often mild and of brief duration. Occurring after crisis even mild symptoms of cardiac failure are serious as they indicate considerable damage of the heart muscle. Cardiac weakness develops as a rule, in cases with severe toxemia, extensive consolidation and hyperpyrexia.

We have already alluded to the high incidence of pericarditis in the post-mortem room as compared to the clinical findings. While a more careful examination of the precordium would undoubtedly bring about a higher percentage of cases of clinical pericarditis, it is to be borne in mind that the pericardial changes are often terminal in character and furthermore, that the confusion of sounds often renders an accurate observation very uncertain.

In a study of the physical signs occurring in the early stages of pneumonia Conner and Dodge determined their frequency as follows: (1) Circumscribed area of feeble and indistinct breathing as compared with the opposite side. (2) Circumscribed impairment without or with tympanitic quality (sitting up). (3) Crepitant râles. (4) Slight increase in intensity and clearness of vocal resonance.

Wilson Fox gives the following sequence of physical signs, corresponding to the anatomical stages of engorgement, hepatization, and resolution.

1. Altered characters of the respiratory sound, which may be weaker or harsher than natural, and attended or immediately followed by fine, crackling râles.

2. Dulness on percussion, attended by bronchial or tubular or suppressed breathing, bronchophony and increased vocal fremitus, together with diminished respiratory movement, chiefly affecting the act of expansion.

3. The return of crepitation, usually in a coarser form; gradual diminution of percussion dulness, together with the return of the respiratory movements and of the characters of the respiration and of the vocal resonance and fremitus to the healthy standard.

The Stage of Resolution.—The duration of this stage varies greatly. Rarely there may be a restoration to the normal within two or three days after the crisis. In the great majority of cases three or four weeks or even longer must elapse before physical signs indicating a normal lung are obtained. In a series of 40 cases in which resolution was unduly delayed McCrae found that the duration extended to the fourth week in 5 cases, fifth week in 10 cases, sixth week in 4 cases, ninth week in 3 cases, and tenth, eleventh and twelfth weeks each 1 case. The gradual disappearance of the consolidated area can be observed from day to day. If no such change is apparent one should be alert to the possibility of an abscess, gangrene or the development of an effusion. The former two conditions will generally manifest themselves sooner or later, by typical symptoms. The presence of an effusion is more apt to be overlooked. One not infrequently sees cases in which it is stated that resolution has never taken place and that the signs of consolidation persist. As a rule one of two things has occurred: either the pneumonia has been complicated by an effusion or, as occasionally happens, an attack of dry pleurisy, followed by an effusion, was ushered in with chilly sensations, fever and pain in the side thus simulating lobar pneumonia. Rarely the affected portion gradually undergoes fibroid changes. Absorption of the exudate and the restoration of the lung to its normal state is the rule; therefore in any case in which the physical signs show no change one should always bear in mind the possibility of one of the above changes being present and by means of an exploring needle and the X-rays determine the exact nature.

Relapse.—There is considerable confusion as to the meaning of this term. Its use should be restricted to those rare instances in which a few days after the subsidence of the original attack a fresh invasion of the lung tissue occurs. "If the lung, after an ordinary croupous pneumonia involving one or several lobes, becomes normal after the fever has terminated by crisis or lysis, the patient is convalescent; and if at least three days or several weeks after the deferescence a new infiltration of the same or other lobes with all the characteristic phenomena of a local and general nature occur, a relapse has without question taken place" (Wagner).

Among 5966 cases collected from the literature a relapse occurred in 36 or 0.60 per cent. (Norris).

Clinical Varieties of Pneumonia.—Pneumonia is not infrequently encountered in forms which present more or less well-marked variations from the ordinary frank type of the disease. In some instances nothing more is meant than that certain symptoms predominate; in others the variation has reference to the character of the anatomical lesion. In the recent influenza epidemic atypical forms of pneumonia were encountered with great frequency. (See section on Influenza.)

Asthenic Pneumonia.—This term is applied to pneumonias characterized by extreme prostration. The onset is often insidious. Physical signs may be absent and this is also true of the subjective symptoms. In other instances there are severe nervous symptoms such as delirium, great prostration and finally coma. Asthenic pneumonia is a common occurrence in the aged and in those enfeebled by chronic illness.

Senile Pneumonia.—This is essentially the same as asthenic pneumonia. The disease as seen in those of advanced years very commonly has an insidious onset, ill-defined physical signs and severe constitutional symptoms.

Terminal Pneumonia.—In those who are the victims of some chronic affection such as diabetes, cardiac, renal or pulmonary disease the final act of the drama may be an attack of pneumonia. Terminal pneumonias are common during the winter months and like the asthenic and senile types are often characterized by a paucity of physical signs and marked prostration.

The three types above mentioned are not infrequently overlooked because they lack the characteristic features of frank pneumonia both as regards the physical signs and the symptoms. The occurrence of respiratory symptoms no matter how slight or evidences of prostration in the aged and in those suffering from some chronic ailment should always suggest the possibility of lobar pneumonia.

Pneumonia in Alcoholic Subjects.—In this type the cerebral symptoms are frequently so marked as to suggest delirium tremens. The respiratory symptoms may be very trivial. It is currently believed that in alcoholics the disease affects the apex of the lung more frequently than the base.

Typhoid Pneumonia.—Mention is made of this term simply to caution against its use. The designation is misleading as one is never certain whether it has reference to a patient who has passed into the typhoid state or whether the pneumonia has been associated with typhoid fever.

Larval or Abortive Pneumonia.—Occasionally cases are seen in which the disease runs a course of from one to three days. In such cases the ymptoms may be quite characteristic of pneumonia but the physical ndings are, as a rule, inconclusive. Abortive pneumonia is most apt be encountered in institutions in which the disease is for the time revailing.

Epidemic Pneumonia.—The fact that pneumonia may assume the ortions of a mild epidemic has already been alluded to in dealing with he etiology of the disease.

Post-operative pneumonia has been referred to under etiology.

Central Pneumonia.—In those cases in which the symptoms point onclusively to lobar pneumonia but in which physical signs are very light or wanting, the lesion is deep-seated or centrally placed. This nay continue throughout the disease or after three or four days the esion may extend sufficiently near the periphery of the lung to admit f its recognition.

Apex Pneumonia.—When lobar pneumonia affects the upper lobes t is believed to be of a more severe type. Prostration, hyperpyrexia, and cerebral symptoms are often marked. In children and in alcoholics the apex is very commonly involved. The right apex is affected about twice as often as the left.

Wandering or Creeping Pneumonia.—Practically every case of pneumonia spreads to some extent. As a rule, it extends from a given spot but limits itself sharply to one lobe. In some cases, however, it slowly extends spreading from lobe to lobe by contiguity; in others the process apparently develops afresh at different points in the same or even the opposite lung.

Massive Pneumonia.—This is a rare form in which the large bronchi become entirely filled with the croupous exudate. Owing to the blocking of the bronchi the physical signs may be limited to marked flatness on percussion such as occur in pleurisy with effusion. Both the breath and voice sounds are absent.

Double Pneumonia.—Both bases may be affected simultaneously or the apex of one lung and the lower lobe of the opposite lung may be involved.

Pneumonia in Children.—Lobar pneumonia as seen in young children often presents well-marked differences from the disease as it occurs in adults. Instead of the initial chill the disease may be ushered in with an attack of vomiting, occasionally by a convulsion, or the child may be listless and refuse to eat for several days prior to the onset of the fever. The temperature rises rapidly and is usually higher than that seen in dults, often reaching 105° to 107°F. Nervous symptoms are usually rominent, the child becoming delirious and unable to recognize anyone; ater the delirium is succeeded by drowsiness and semiconsciousness. Vomiting which may be severe and persistent is often a marked feature f the disease in children. These symptoms at first sight suggest some erebral affection especially meningitis but meningitis rarely has so acute n onset.

Occasionally a child ill with pneumonia develops an erythematous sh which bears a superficial resemblance to scarlet fever. It differs om the rash associated with the latter in that it is not punctate and is brief duration.

The disturbance of the pulse respiration ratio (normally 4.5 to 1) is very suggestive. Expectoration is rare in children as they usually swallow the sputum.

The apex of the lung is affected somewhat more frequently than the lower lobe. The physical signs may for a few days be entirely absent or very inconclusive. Northrup has epitomized the physical findings as follows: Absent or diminished respiratory murmur over one lobe or portion of a lobe; râles of any kind, perhaps appearing only late, either in a localized shower or diffusely scattered; broncho-vesicular breathing—a bronchial whiff; slight dulness, becoming marked only late; and, lastly, cough.

Mason[1] in a study of the X-ray findings in lobar pneumonia in young children found that the consolidated area was triangular or conical in shape with the base at the periphery of the lung. The consolidation always started at the periphery and extended in toward the hilus, and the base of the consolidated area is very frequently situated at a point corresponding to the apex of the axilla. It is well known that lobar pneumonia as it occurs in children may be attended with nothing more than slight impairment of the percussion note, bronchial breathing and voice being absent. In the ~~absence of the~~ latter signs Mason states that the pneumonic process does not extend to the hilus. This leaves an area of normal air tissue which dissipates the bronchial quality of the breath and voice sounds which have their origin in the trachea and large bronchi at the root of the lung. Absent or slight physical signs are due therefore to peripheral rather than a central location of the pneumonic process.

Secondary Pneumonia.—This form is met with chiefly as a complication of one of the specific fevers particularly diphtheria, typhoid fever, typhus and influenza. The symptoms are often indefinite and the physical signs of equivocal import. The symptoms are apt to lack the striking features of frank croupous pneumonia and the physical signs rarely amount to more than impaired resonance, feeble breathing and a few crackling râles. One is often in doubt as to whether there is a hypostatic congestion, edema, pulmonary infarct, a central pneumonia or a broncho-pneumonia.

A disturbance of the normal pulse-respiration ratio—4.5 to 1—is always suggestive and should lead to careful physical examination. In spite of precautions the lesion is often overlooked.

Diagnosis of Pneumonia.—The recognition of a typical case of pneumonia is in the great majority of instances, an easy matter. A history of a chill followed by pain in the side, fever, rapid respirations, the presence of herpes, a leukocytosis and bloody or rusty sputum are of themselves sufficient to warrant a diagnosis, even in the absence of physical signs. The latter it is to be recalled, may not be present for a few days and in some cases are never definitely determined. When present the physical signs taken in conjunction with the symptoms render the diagnosis practically certain. The evolution of the physical signs has been considered in full and need not be considered further.

In the great majority of cases of atypical pneumonia the disease occurs in the aged, in those debilitated by some chronic disease and in children. In such cases, while the patient is usually very ill, there is a paucity of both physical signs or symptoms or the symptoms while very severe are ot such as occur in typical pneumonia. One should always recall that nder these circumstances pneumonia is always a possibility and every eans should be taken to determine its presence. It is in such cases that lterations in the pulse-respiration ratio sometimes furnish the clue. leukocytosis also points to pneumonia although in very severe cases leukopenia may be present. If expert laboratory assistance is available, a blood culture should be made in doubtful cases as this is the most ertain method we possess.

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[1] *Amer. Jour. Diseases of Children*, March, 1916.

The conditions which are most likely to be confused with lobar pneumonia are acute *tuberculous pneumonia* or pneumonic phthisis, *broncho-pneumonia, pleural effusion, pulmonary congestion, pulmonary infarct and atelectasis.*

Acute Tuberculous Pneumonia.—Acute pneumonic phthisis is considered in full in the section dealing with tuberculosis. It is sufficient to remark here that tuberculosis often first manifests itself with a chill, pain, cough and signs of consolidation. In some instances it is impossible for a week or more to distinguish it from lobar pneumonia. At the end of ten days or two weeks, however, the temperature changes from the continuous to the remittent or intermittent type and the sputum becomes purulent in character. While pure bronchial breathing may be heard over the consolidated area the absence of breath sounds is not unusual and is of considerable diagnostic importance. In every case of protracted pneumonia the sputum should be examined for tubercle bacilli.

Acute tuberculous pneumonia usually involves one of the upper lobes nd in the majority of cases occurs in an individual whose health has een bad or who has had previous pulmonary trouble. The occurrence f an hemoptysis points to tuberculosis rather than pneumonia.

Rarely true lobar pneumonia occurs in a definitely tuberculous invidual. A young woman with a moderately advanced tuberculous sion at the right apex which had become arrested, was suddenly seized ith a chill, pain in the right side and cough. The temperature rapidly se to 103°F. and the respirations to 30. At the end of two days a all area of dulness and bronchial breathing was heard near the angle the scapula; this gradually spread and involved all of the right lower be. The attack ran a course of two weeks, the temperature falling by sis. In the course of the next three months the lower lobe gradually as restored to normal. There was no lighting up of the tuberculous rocess. In this case it was impossible to tell during the attack or indeed or some weeks afterward, whether the process was a true pneumonia or a widespread tuberculous infiltration.

Broncho-pneumonia.—There is no difficulty in distinguishing between typical lobar pneumonia and broncho-pneumonia but in those of advanced years and in the debilitated croupous pneumonia not infrequently s mistaken for the broncho-pneumonic form. The principle features of roncho-pneumonia are a gradual onset and a much longer duration han occurs in lobar pneumonia. Dyspnea and cyanosis are more marked han in the lobar form and the temperature is lower and subject to ore marked intermissions. Both lungs are affected and the physical xamination shows a preponderance of signs of bronchitis over those of onsolidation.

Pleural Effusion.—At first sight the distinction between a pleural fusion and lobar pneumonia should be easy. As a matter of fact the mistaking of the one for the other is not uncommon. In some instances the differentiation is not easy and one cannot be certain without having recourse to the exploring needle, a procedure which should always be followed in doubtful cases. In the typical case of pneumonia there is bronchial breathing and increased vocal and tactile fremitus while in pleural effusion the breath sounds are absent or very distant and the fremitus, both vocal and tactile, is absent. It not infrequently happens, however, that the signs peculiar to one may occur in the other. Thus distant, almost inaudible, breath sounds may occur in pneumonia while over an effusion loud bronchial breathing may be heard. Ægophony over the upper limit of an effusion is considered peculiar to this condition but it may occur over consolidation as I had occasion to note only recently. In large effusions which fill one-half or two-thirds of the pleural cavity little difficulty is experienced as the bulging of the affected side, the obliteration of the interspaces, the lack of motion and the displacement of the apex beat of the heart renders a diagnosis possible in most cases by inspection alone. It is the small effusion in which displacement of the thoracic viscera does not occur, that makes the diagnosis difficult. The presence or absence of Grocco's triangle is often of service. The exploring needle or X-rays should always be resorted to in case of doubt.

There is another phase of the subject which should be mentioned, namely, the development of an effusion in association with the pneumonic process. As a rule such effusions are purulent in character; in children they are practically always so. Generally no suspicion arises as to their presence until after the stage of resolution has set in. It then becomes apparent that the affected lung is not clearing up and that the physical signs persist. The diagnosis rests partly on symptoms and partly on physical signs. If the temperature has fallen by crisis there is a secondary rise two or three days later and the respiratory rate either fails to fall or becomes increased. If the temperature falls by lysis, a secondary rise may occur before the normal has been reached.

There may be absence of breath sounds but as the fluid has formed over consolidated lung the breathing may be bronchial. If the effusion gradually increases in size, bulging, lack of motion and displacement of the viscera take place. It is to be borne in mind that an empyema, sometimes loculated, is not infrequently the cause of symptoms and physical signs ascribed to delayed resolution.

Pulmonary Congestion.—Very often patients who have been confined to bed because of some protracted illness such as typhoid fever, develop varying degrees of congestion in the posterior portions of the lungs, especially at the bases. The condition is always bilateral. The breathing may be somewhat suppressed but is otherwise normal and the percussion note is rarely more than slightly impaired. Very often, however, fine crepitating râles are heard which are indistinguishable from those occurring in pneumonia. Often they are transient and disappear after a few deep breaths but they may persist. The fact that the condition is bilateral and the knowledge that it is not uncommon in those long confined to bed should serve to distinguish it from pneumonia.

Pulmonary Infarct.—Among those suffering from chronic valvular heart disease the occurrence of a pulmonary infarct is not uncommon. The onset is abrupt, due to plugging of one of the pulmonary vessels by an embolism. The patient is usually seized suddenly with pain in the side, shortness of breath, and later there is cough and the expectoration of bloody sputum. On physical examination there may be a small area of crepitating râles with or without dulness on percussion and bronchial breathing. In some instances the physical signs closely resemble those obtained in pleurisy with effusion. The temperature is usually low. Fowler states that in cases of infarction the patient will almost certainly be found sitting up in bed while this rarely happens in pneumonia. Examination of the heart should make it possible to determine the existence of a valvular lesion if this is not known already.

Pulmonary infarction as the result of venous thrombosis is more confusing as the pulmonary symptoms may appear before the thrombosis in the vein manifests itself. The continuation, day after day, of frankly bloody sputum associated with physical signs of consolidation in one of the lower lobes is very suggestive of pulmonary infarction due to a venous thrombosis.

Atelectasis or Collapse of the Lung.—Collapse of one of the lower lobes of the lungs may be mistaken for pneumonia. The condition is sometimes seen in association with acute bronchitis, great hypertrophy of the heart or in large pericardial effusions. Pasteur has called attention to massive collapse of one of the lower lobes following surgical operations.

Collapse of one of the pulmonary lobes may occur with the same suddenness and intensity as a pulmonary embolism, but in most instances it is attended by less violent symptoms. The condition is characterized by dyspnea, pain in the chest and the expectoration of viscid sputum. Usually the symptoms subside rapidly, rarely lasting more than 24 hours. If the viscid sputum persists for a longer period it is likely that a bronchitis or broncho-pneumonia has developed in the reëxpanding lung.

Physical examination shows some retraction of the affected side, impairment of the percussion note, weak, tubular breathing, and the absence of vocal fremitus and râles. The conclusive sign of massive collapse of the lung is displacement of the heart's apex beat toward the affected side, and it is the only sign peculiar to the condition (Pasteur).

Occasionally in the early stages it may be difficult to differentiate *typhoid fever* and *meningitis* from the atypical forms of pneumonia. It is in such cases that one should always, whenever possible, enlist the aid of the laboratory. The confusion which sometimes arises between acute lobar pneumonia and acute abdominal inflammations has been alluded to above.

FRIEDLÄNDER'S BACILLUS PNEUMONIA

Etiology.—A very fatal but fortunately a rare form, of pneumonia is that known as Friedländer's bacillus pneumonia. The exciting cause of this type of pneumonia is the bacillus mucosus capsulatus, also known as the pneumobacillus and Friedländer's bacillus. Although this organ- vas identified by Friedländer in 1882 it has never attracted much tion as one of the causes of lobar pneumonia. "Thus far only thirty-three cases of pneumonia have been reported so fully that an reasonably be considered to have been produced solely by the s mucosus capsulatus. Some of these may be questioned as vocal cases of so-called 'Friedländer pneumonia.'"[1]

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[1] SISSON and THOMPSON: *Am. Jour. Med. Sc.*, Nov. 15, 1915.

The bacillus mucosus capsulatus is found either as a primary or secondary invader in about 5 per cent. of all cases of pneumonia. It h been noted also in a considerable number of cases in which the tuberc bacillus is the primary source of infection. Owing to the fact that th diagnosis rests entirely on the bacteriological findings it is quite possibl that Friedländer's bacillus pneumonia is of more frequent occurrence tha the number of reported cases would indicate.

The organism may be found in pure culture in the sputum or in th blood, the latter being the most conclusive proof.

This type of pneumonia occurs chiefly in late adult life; it is rarel encountered in infants and young children.

Morbid Anatomy.—A lung which has become the seat of pneumoni due to the Friedländer bacillus presents certain characteristics whic serve to distinguish it from infections due to the pneumococcus. Th cut surface lacks the granular appearance so characteristic of pneu mococcus infections. In the early stages the process is more or les lobular in its distribution and hemorrhagic foci are not uncommon. Th gives the lung a mottled, marble-like appearance. Later the lobula areas tend to coalesce, forming a more or less homogeneous appearanc The lung then has a grayish-slaty color. Areas of necrosis and absces formation are not uncommon and are more frequently seen in this for of pneumonia than in pneumococcus pneumonias. Practically the sam changes have been demonstrated in experimental animals by Sisson an Walker.[1] These observers found that the lungs of cats infected wit the bacillus mucosus capsulatus showed in the early stages a lobula distribution, which later became homogeneous, and the presence hemorrhagic foci. The lung was at first mottled and marble-like i appearance, later it became of a grayish color and the cut surface wa not granular.

The exudate from the lung is abundant and of a slimy, mucoid appea ance. A fibrinous exudate is nearly always present on the pleura an this may lead to a serofibrinous or purulent effusion. Occasionally th other serous membranes are involved.

Histologically the alveoli are seen to be filled with a serous exuda containing red blood cells, pus cells and desquamated epithelial cell Fibrin is less abundant than in the ordinary type of pneumonia. striking feature of the disease, in most instances, is the enormous numbe of the infecting organisms which may be both extracellular an intracellular.

Symptoms.—Aside from the extreme severity of the constitution symptoms and the brief duration of the disease there is nothing clini ally to distinguish this type of pneumonia from the ordinary form. I more often begins without a chill than with one. Usually the ons is with pain in the side, cough and dyspnea. Delirium may occur earl Herpes labialis so frequently seen in the pneumococcus form rarely occu in this type. The evidences of a severe toxemia are marked and wea ness, coma, and cardiac failure may appear early in the course of th disease. The temperature may be continuous or remittent and as a ru is not as high as that seen in the ordinary form and the pulse rate is al apt to be lower. Considerable importance is attached to the charact of the sputum. This is abundant, bloody, very slimy, non-purulent character and contains great numbers of the infecting organism. A leukocytosis is commonly present.

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[1] *Jour. Exp. Med.*, Dec. 1, 1915.

This form of pneumonia is usually of brief duration and a fatal termination is the rule. Death may occur in a little over 24 hours from the onset of the initial symptoms.

Physical Signs.—There is nothing to distinguish this type from the usual form of pneumonia so far as physical examination is concerned.

Diagnosis.—Pneumonia due to the bacillus mucosus capsulatus may be suspected in a patient with signs of pulmonary consolidation, severe toxemia and the presence of an abundant, sanguineous, and slimy sputum. The definite proof, however, rests on the occurrence of large numbers of the Friedländer organism in the sputum or in blood cultures.

PSITTACOSIS

The term psittacosis (from ψιττακος, parrot) is applied to an infectious disease transmitted to man by parrots and characterized by a typhoid state and atypical pneumonia. In parrots the disease is distinguished by listlessness, diarrhea, and loss of appetite. The feathers stand on end, the wings droop, and marked wasting and debility develop rapidly. The disease is almost invariably fatal in birds. Nocard, in 1893, isolated from diseased birds a bacillus which has many of the the cultural characteristics of the bacillus typhosus. This organism is accepted as the infecting agent in birds although it has been found in man in but a single instance.

Etiology.—Although several small house epidemics had been described prior to the Paris epidemic in 1892 and 1893, it was not until the occurrence of the latter that the disease attracted attention.

The Paris epidemic followed an importation of parrots from South America. There were 49 cases, with 16 deaths, in 1892 and the following year 7 more cases were observed with 5 deaths. Since that time a number of small house epidemics have been noted in France, Italy and Germany. In this country 3 probable cases of psittacosis have been reported by Vickery and Richardson.[1] Up to the present time about 100 cases have been recognized.

The disease is believed to be transmitted from bird to man and never from man to man. The infection may occur as the result of feeding from mouth to beak, a practice followed by pigeon raisers and which in the latter sometimes causes infection with the aspergillus. More commonly the infection is acquired by fondling the sick bird or by cleaning the contaminated cages.

Although the cases reported seem to indicate that there is a close relationship between the disease occurring in parrots and that which occurs in man the proof is not conclusive. In the first place the organism isolated by Nocard and believed to be the infecting agent in parrots, has been found but once in human beings nor have agglutinating tests been uniform; and in the second place, relatively few individuals acquire the infection although ample opportunity is offered in the case of bird fanciers and those who have parrots for pets. Within the past few years there has been no reference to the disease in the literature.

[1] *Trans. Assoc. Am. Phys.*, 1904, p. 364.

Morbid Anatomy.—In parrots dying from the disease, the pathological findings consist of a severe enteritis and cloudy swelling of the spleen, liver and kidneys. Nocard's organism is found in the marrow of the wing bones.

In man, little is known of the pathology. Cases have been recorded in which a lobar or lobular type of pneumonia was found. Cultures from the heart's blood, the lungs and other organs have shown the presence of the pneumococcus, streptococcus and other organisms. Failure to find Nocard's organism is explained on the ground that this organism has been killed off by secondary invaders.

Symptoms.—Dieulafoy places the incubation period at from seven to twelve days, while other observers state that it may last for as long as twenty-five days.

The disease may be ushered in abruptly or it may have an insidious onset. When the onset is sudden the clinical picture is not unlike that of pneumonia. There is a chill, marked prostration, and fever which rises rapidly to 102° or 104°F. and is continuous in type with slight daily remissions. Cough appears early and is usually attended with mucoid, rusty or bloody sputum. In some cases there is no sputum. Herpes are absent.

In other cases the onset is insidious and bears a striking resemblance to typhoid fever. The patient complains of headache, malaise, weakness, anorexia and diarrhea or constipation, usually the latter. The breath is foul and the tongue heavily coated. A petechial eruption is sometimes present. The spleen has been noted as being enlarged in some cases. Delirium, apathy or stupor is often present. Respiratory symptoms are also present in the typhoid type of the disease.

A severe stomatitis or peribuccal edema may be an early manifestation in those who have practised mouth to mouth feeding.

The disease remains stationary for from eight to ten days when the temperature falls, usually by lysis. Convalescence is, as a rule, protracted. The total mortality for reported cases is about 30 per cent.

Physical Signs.—Examination of the chest may show the presence of fine moist râles throughout both lungs or physical signs indicative of either a broncho-pneumonia or lobar pneumonia. Often the physical findings are inconclusive.

Diagnosis.—This rests entirely on a knowledge of the exciting cause. If it is known that the patient has been in contact with a diseased parrot, psittacosis should at once suggest itself. Especially should one's suspicions be aroused if several members of a household are simultaneously affected. In the absence of such knowledge the case is certain to be regarded as one of atypical pneumonia.

BRONCHO-PNEUMONIA

This form of pneumonia is also known as catarrhal pneumonia, lobular pneumonia and capillary bronchitis. The latter term should never be used. While a lesion limited to the finer bronchioles is possible theoretically, practically it never exists alone, as an inflammatory process involving the smaller bronchi inevitably extends to the anatomically related or contiguous vesicles as well. The term broncho-pneumonia is much the most preferable designation and by common consent is the one now in general use. As the name implies the process involves both the bronchi and the pulmonary vesicles.

Inasmuch as broncho-pneumonia plays such an important part in influenza, and, when associated with the latter disease, displays such marked variation, both pathologically and clinically, the section on influenza should be read in connection with this.

Etiology.—The disease occurs as a primary and secondary manifestation and is encountered most frequently at the two extremes of life. The two sexes are about equally affected. The primary form is almost invariably seen in children under two years of age. Beyond this age period the disease is usually a secondary manifestation. Statistics show that in about one-third of all cases the disease is primary.

As a secondary manifestation broncho-pneumonia occurs under a variety of conditions.

(*a*) As a complication of or sequel to one of the acute infectious diseases broncho-pneumonia is encountered with great frequency. While any of the acute infections may give rise to broncho-pneumonia the infections most to be feared are diphtheria, measles and whooping cough. There is a very widespread belief among the laity that measles and whooping cough, especially the latter, are relatively harmless affections. For this reason children are often purposely exposed to these diseases in order that what is believed to be a necessary episode in their lives may be over and done with. Although both these diseases are relatively harmless in themselves it cannot be too strongly insisted upon that they are extremely dangerous because of their complications and sequels. A very considerable proportion of deaths from broncho-pneumonia occur in children in whom the primary infection was either measles or whooping cough. A very severe form of broncho-pneumonia following an attack of measles in young adults occurred in several of the army cantonments during the winter of 1917–18. The condition was remarkably infectious and required strict isolation to prevent its spread. The infecting organism was the streptococcus hemolyticus which has been shown to be present in the tonsils of many individuals. Empyema was a frequent accompaniment.

(*b*) In both children and adults broncho-pneumonia may succeed an attack of acute bronchitis, the inflammatory process extending along the bronchial tubes and eventually implicating the air vesicles. A secondary broncho-pneumonia may arise also in cases of bronchiectasis, chronic interstitial pneumonia, pulmonary abscess or as a result of an empyema rupturing into the lung. In like manner broncho-pneumonia may occur in cases of chronic tuberculosis with cavity formation, the infecting material from the cavity giving rise to an ordinary broncho-pneumonia, a tuberculous broncho-pneumonia or both.

(*c*) A focus of infection anywhere in the body may by metastasis give rise to a septic broncho-pneumonia. Among such conditions may be mentioned acute otitis media, suppurative appendicitis, salpingitis, infections of the urinary tract, etc.

(*d*) Broncho-pneumonia is to be classed among the most frequent of the terminal infections. In the aged and debilitated and in those suffering from some chronic affection such as heart disease, nephritis, malignant disease, diabetes, etc., it is one of the most frequent causes of death.

(*e*) Still another form of the disease is the so-called inhalation or deglutition pneumonia. In health the sensitiveness of the larynx prevents the passage of food and drink into the trachea and bronchi. If, however, the larynx becomes insensitive as the result of the narcotic effect of alcohol, ether or chloroform or as the result of stupor or unconsciousness, such as occurs in uremia, apoplexy, etc., food or drink or septic material may be aspirated into the lungs and set up a very severe form of broncho-pneumonia (Fig. 276).

Aspiration pneumonia may also follow the inhalation of material from a bronchiectatic cavity, a tuberculous cavity or it may occur as a sequel to a large hemoptysis. This form of the disease not infrequently follows operations about the nose, mouth or larynx.

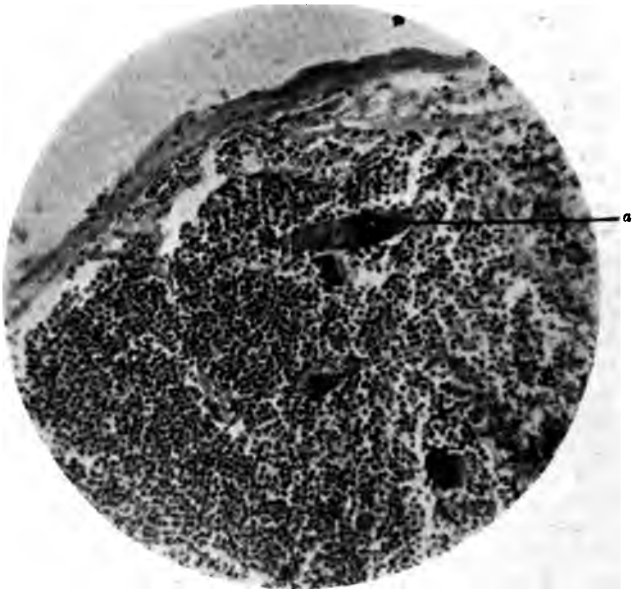


FIG. 276.—Meat fibers (*a*) broncho-pneumonic focus. (*Dr. F. D. Weidman.*)

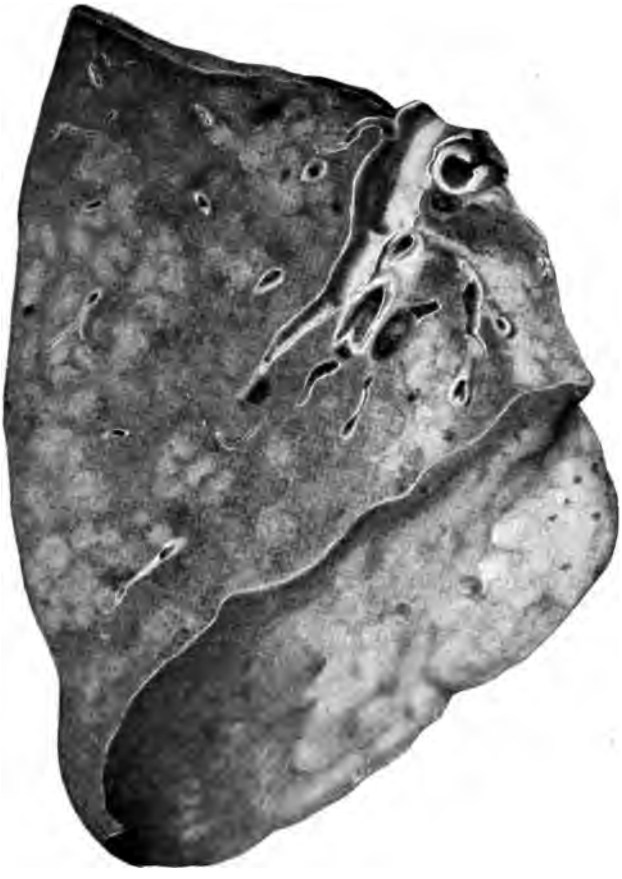
Children who are debilitated as the result of syphilis, rickets or diarrhea are extremely subject to broncho-pneumonia, and children living amidst insanitary surroundings and inmates of orphan asylums or foundling homes are especially prone to develop the disease.

Inasmuch as broncho-pneumonia frequently develops as a sequel to acute bronchitis the disease is usually more prevalent during the winter and early spring months. At this time respiratory infections are far more common than at other seasons of the year and in this respect broncho-pneumonia is no exception.

Morbid Anatomy.—The affection is nearly always bilateral, although the lesions may be much more extensive on one side than the other. Holt found the disease bilateral in 82 per cent. of cases coming to autopsy. The disease has a decided preference for the posterior aspect of the lower lobes. The middle lobe of the right lung is the least affected portion.

A lung the seat of a broncho-pneumonic process is heavier than normal, congested and while crepitant throughout, reveals to the touch ed nodules of consolidation (Fig. 277). Pleurisy is less common in oncho-pneumonic form than in the lobar type of the disease. the lesion is just beneath the pleura the latter may be slightly d or there may be a deposit of fibrin or purulent material. Oc-ally a moderate-sized effusion may occur.

very common finding, especially in young children, are areas of asis. The most frequent sites of the collapsed vesicles are the



277.—Lobular pneumonia in adult lung, showing patchy areas of consolidation. (*MacCallum's Pathology.*)

ior inferior margins of the lower lobes, the free margins of the lung rly and at times the inner margin of the lungs adjacent to the The atelectatic area is sharply defined, shrunken, depressed below rface and of a bluish or dark violet tint. On section it is smooth istening in appearance and on pressure a small amount of bloody exudes. The tissue not only feels airless but also sinks when placed ter. About the collapsed areas the pulmonary tissue may be tous or show compensatory emphysema. On section the broncho-pneumonic process shows an intense congestion of the bronchial mucous membrane and small groups of vesicles. The smaller bronchi and bronchioles and the affected air cells are completely filled with an inflammatory exudate which contains little or no fibrin. The exudate consists mainly of serum, a few red cells, numerous leukocytes and large mononuclear or catarrhal cells.

The patches of consolidation vary greatly in size. They may consist of small foci the size of a pinhead or be as large as a hazelnut. A number of distinct foci may merge into each other producing a large area resembling croupous pneumonia. Occasionally in young children a fulminant type of the disease occurs in which death occurs before consolidation becomes evident.

On section of the lung the broncho-pneumonic area is seen to project slightly above the cut surface and is more or less sharply circumscribed. At first the lesion is firm and reddish in color but in its later stages it is reddish gray, gray, or yellowish in color and very friable. On pressure a turbid blood-stained fluid can be expressed in which may be seen purulent material.

In the aspiration forms of the disease numerous small abscesses may develop at the site of the lesions. When the disease runs a protracted course the tubercle bacillus is to be suspected. In children a widespread tuberculous broncho-pneumonia is not an uncommon finding. In fatal cases abscess formation is not infrequent but gangrene is rare. When recovery takes place the consolidated areas undergo complete resolution in the great majority of cases. In some instances a rather diffuse bilateral fibrosis and bronchiectasis follow an attack of broncho-pneumonia.

Bacteriology.—Mixed infections are almost the rule in broncho-pneumonia, the streptococcus being the most constant organism. Among the organisms which have been found alone or in association with others may be mentioned the pneumococcus, streptococcus, staphylococcus aureus and albus, Friedländer's bacillus, the influenza bacillus, the diphtheria bacillus, etc. Prior to the recent epidemic of influenza the teaching was that neither the symptoms nor the lesions in the lungs showed any marked differences as the result of infection by different organisms. Our experience with influenza, however, has shown that the disease does vary in accordance with the infecting organism. Thus streptococcic broncho-pneumonias are apt to be characterized by symptoms of a general septicemia. Pathologically certain organisms, notably the streptococcus and the influenza bacillus, produce marked interstitial changes, constituting what has been termed an interstitial broncho-pneumonia. In still other instances the broncho-pneumonic areas become confluent and involve a part or all of one lobe, thus producing an atypical form of lobar pneumonia. (See section on Influenza.)

Symptoms.—The recognition of broncho-pneumonia is not easy because of the varied clinical picture. Then, too, in infants the disease is, at times, so violent that death may ensue in from 12 to 24 hours, and before the true nature of the trouble is appreciated. In marked contrast is the insidious onset and the paucity of both symptoms and physical signs which so frequently characterize the disease in the debilitated and the aged.

In the *primary form* of the disease which occurs almost exclusively in children under two years of age, the onset is usually sudden with a chill or convulsion. The temperature rises rapidly and is usually continuous in type. In many cases cerebral symptoms play a prominent part and may dominate the picture leading to a diagnosis of meningitis. The child is peevish, very restless, easily excited, is sleepless and often very delirious. The disease often terminates by crisis. The mortality from primary broncho-pneumonia is slight. Owing to the presence of physical signs indicating consolidation of the lung the condition is often mistaken for lobar pneumonia.

In the *secondary form* both the onset and course of the disease are essentially different. Instead of a sudden onset in a child previously well there has been present an acute bronchitis either alone or as a complication of one of the acute infections, particularly measles or whooping cough. The transition from the minor to the serious affection is gradual and the first thing to arouse suspicion as to the presence of broncho-pneumonia may be the increase in the respiratory rate and an accession of fever. In a child convalescing from measles or whooping cough the occurrence of these symptoms and the presence of râles in the lungs, even without evidence of consolidation, are sufficient to warrant a diagnosis of broncho-pneumonia.

The *fever* is usually moderately high and may be either remittent or intermittent in type. Usually the difference between the morning and evening temperatures amounts to 3°F. or more (Fig. 278). Occasionally there may be hyperpyrexia while in weak and debilitated children the temperature may be but slightly elevated or even subnormal. In secondary broncho-pneumonia the temperature always falls by lysis, the febrile period lasting from one to three weeks.

The *pulse* is usually very rapid but the increase in frequency is relatively less than that of the respiration. If the embarrassment of the right heart is marked the pulse rate runs up to 150 or higher and is apt to be irregular.

The *respiratory rate* is markedly increased and in severe cases the intense dyspnea may be the most striking feature of the disease. A respiratory rate of 50 or more is not uncommon. The difficulty in breathing is usually in proportion to the extent of the pulmonary involvement. In fatal cases marked dyspnea is frequently associated with atelectasis. In addition to the respiratory distress deficient aeration of the blood is shown by the cyanosis of the face and finger tips and the anxious expression. In the severe fatal cases the child rapidly passes into a condition of asphyxia.

Pain is rarely a marked feature in this form of pneumonia and in many cases is entirely absent. It usually occurs in the form of a dull aching sensation over the site of the pulmonary lesion.

Cough is usually a prominent feature and is often very distressing. A vigorous cough indicates absence of toxemia while a feeble cough or the cessation of cough is usually associated with severe cases. In children
tum is usually swallowed. In adults it may consist of a thin
or it may be mucopurulent in character. Occasionally the sputum
d streaked.

Skin.—In severe cases with extensive pulmonary lesions there
lly some evidence of cyanosis due to the embarrassed right heart.
cases the tips of the ears, the lips and the finger tips are of a
ue. When cyanosis is present, the skin is apt to be cool and

clammy. Ordinarily the skin feels hot and slightly moist. Sweati often occurs during the height of the disease. Herpes labialis so co monly met with in the lobar form of pneumonia are rarely seen broncho-pneumonia.

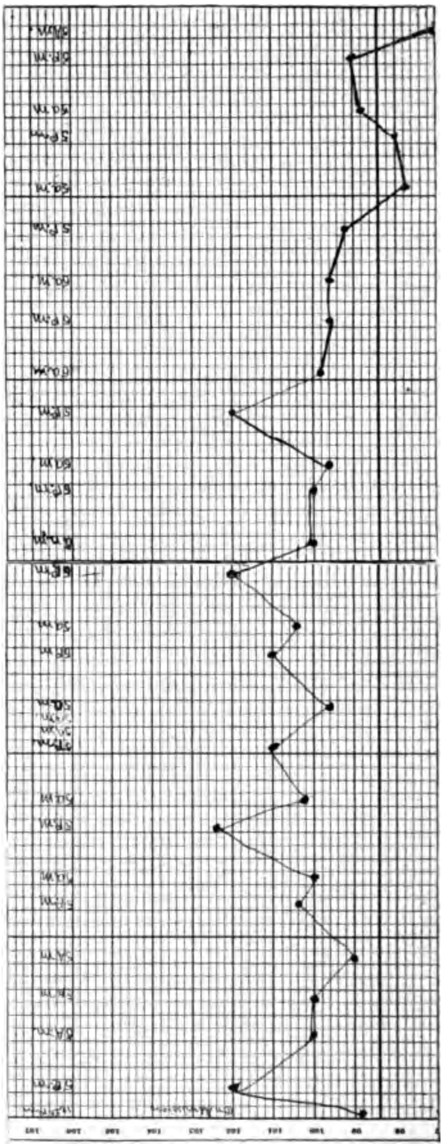


FIG. 278.—Temperature curve in broncho-pneumonia.

Gastro-intestinal symptoms are common. A child ill with bronch pneumonia usually refuses food. Thirst is marked. Vomiting is common symptom and in addition there is often diarrhea, the sto being green in color and containing mucus and undigested food.

Nervous symptoms are often very marked especially in severe cases. The child is restless, irritable, sleeps poorly and at times is delirious.

The *urine* presents the usual characteristics seen in fever patients. It is scanty, high-colored, shows a trace of albumen and there also may be tube casts.

In adults broncho-pneumonia is less common than the lobar form. It is always secondary and presents the same clinical picture as that just given for children.

The *duration* of an attack of broncho-pneumonia is variable. We have alluded already to the fulminant type in which death may occur within 48 hours. In the average case the disease runs its course in from two to three weeks. It may terminate, however, in a week or it may be prolonged beyond three weeks. In the protracted form described by Holt the disease may last for four or five weeks or even longer. In this type of the disease the fever persists but is subject to exacerbations and remissions. The child wastes, becomes anemic, profoundly prostrated and finally dies from slow asthenia. The clinical picture is strikingly similar to tuberculous broncho-pneumonia and the distinction between the two is not easy.

Broncho-pneumonia as it occurs in the aged and debilitated very often begins insidiously and neither the symptoms nor the physical signs are distinctive. As a result the disease often escapes detection during life and is first discovered at the autopsy.

During the winter and spring months when pulmonary diseases are most common even trivial respiratory symptoms are to be viewed with suspicion when occurring in those of advanced years and in those debilitated from some chronic disease. The most significant symptoms are fever, cough and dyspnea, especially in one who is already suffering from bronchitis. Physical examination may show the presence of a diffuse bronchitis with or without patches of consolidation. In some instances there is an absence of symptoms; in others it may be impossible to detect any evidence of a pulmonary lesion and in still others, both symptoms and signs may be so indefinite as to entirely escape notice. A knowledge of the frequency with which broncho-pneumonia occurs as a terminal infection in the aged and debilitated should make one alert in the presence of even the most trifling respiratory symptoms.

An extremely fatal form of broncho-pneumonia is that in which the exciting organism is the *Bacillus pestis*. During a plague epidemic the pneumonic type of the disease is encountered in a small percentage of cases. The infection may be primary due to the inhalation of plague bacilli which have sprayed into the air by coughing or it may occur secondarily to a focus elsewhere in the body.

This form of broncho-pneumonia is characterized by an abrupt onset with a chill, fever which may be continuous or remittent, dyspnea, cyanosis, cough and the expectoration of mucopurulent, blood-streaked, or hemorrhagic sputum.

The physical signs are those indicative of patches of consolidation lower lobes posteriorly. Physical examination also shows marked ement of the spleen.

ysical Signs.—The physical signs of broncho-pneumonia do not hemselves readily to description. If, however, one keeps in mind the pathology of the disease, the irregularity of the physical findings is more easily understood. In children and to some extent in adults, this is facilitated by following the classification of Holt who describes:

(1) The stage of congestion without consolidation. (2) The stage of partial consolidation. (3) The stage with areas more or less completely consolidated. (4) The prolonged form.

First. THE STAGE OF CONGESTION.—*Inspection.*—The breathing is rapid and shallow, the lower intercostal spaces, the submammary region and the epigastrium receding with inspiration. These signs are more marked in infants than in those of mature years.

Palpation.—Palpation is negative except for the occasional occurrence of rhonchial fremitus.

Percussion.—In the early stages of the disease the percussion note may be, and usually is, normal, a fact that Holt has especially emphasized. At the most, there may be a slight area at one base near the spine, in which the percussion note deviates slightly from the normal.

Auscultation.—As a primary infection, the signs are those of a bronchitis affecting the smaller tubes, while if the disease develops from a preëxisting bronchitis of the larger tubes, the râles are of a mixed character. In the beginning the breathing may be normal everywhere, but sooner or later localizing signs in the form of very fine, moist râles (subcrepitant) appear, usually at one or other of the bases. Over this area the breath sounds may then become feebler and higher in pitch. The voice sounds may also become more intense and higher-pitched (see Figs. 279 and 280).

Second. STAGE OF PARTIAL CONSOLIDATION.—*Inspection.*—No change.

Palpation.—Over the area of partial consolidation the tactile fremitus may be increased.

Percussion.—While the auscultatory signs of consolidation are [illegible] to be well marked in this stage, the percussion changes are much less and may be negative. This is ascribed to the superficial nature of [illegible] lesions, and by the fact that the small consolidated areas are separated by normal vesicular tissue. If impairment over a limited area [illegible] been noted previously, however, it is apt to be more pronounced, and [illegible] area of slight impairment considerably extended. Slight changes in the percussion note may be detected at this time over the opposite lung, [illegible] over another portion of the same lung.

Auscultation.—In addition to the auscultatory findings noted in [illegible] first stages, there are added those of more or less complete consolidation, namely, enfeebled breathing, bronchial in character, bronchophony and whispering pectoriloquy.

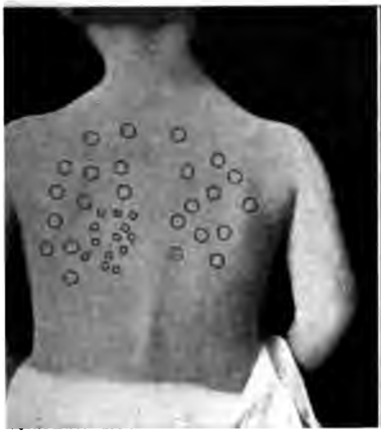
Third. WITH AREAS OF CONSOLIDATION MORE OR LESS COMPLETE.—*Palpation.*—Over the area of consolidation the tactile fremitus is usually increased.

Percussion.—The note is dull but not markedly so owing to the fact that the lesion is superficial and does not extend very deeply into the lung.

Auscultation.—Over the central portion of the consolidated area the breathing is bronchial. As the periphery of this area is approached the breath sounds become less bronchial and in addition râles are heard. The vocal fremitus is increased (see Figs. 281 and 282).

ie signs of consolidation may extend as far forward as the axillary
it they usually stop there.

urth. THE PROTRACTED FORM.—In this type the disease behaves
ee or four weeks as an ordinary case of broncho-pneumonia. Then



!79.—First stage. Coarse rales over
ngs; localized fine (subcrepitant)
the left base. No change in breath-
ıds. (*Holt.*)

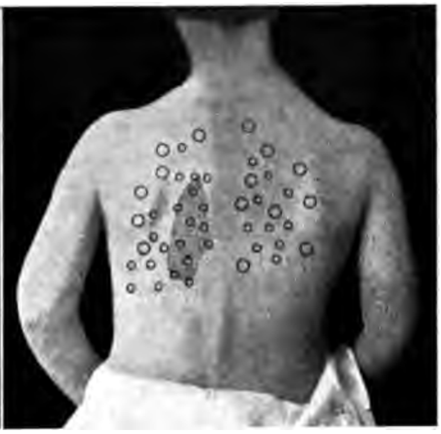
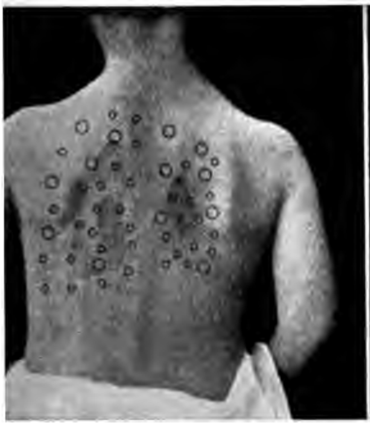


FIG. 280.—Second stage. Coarse and fine râles over both lungs behind; at left base an area of partial consolidation, with broncho-vesicular breathing, exaggerated voice, and very sharp rales. (*Holt.*)



!81.—Third stage. A larger area of
consolidation, and in the center a
rea of complete consolidation, with
al breathing and voice, and slight
Signs over the right lung similar
were previously present over the
Iolt.)

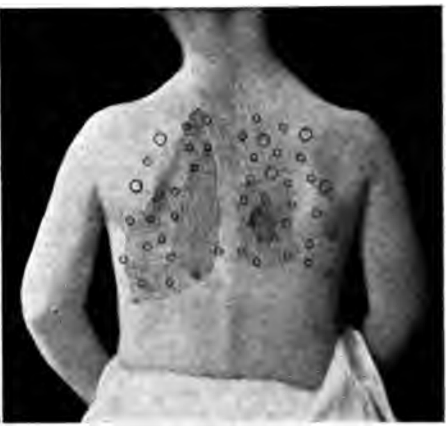


FIG. 282.—Fourth stage. Extensive disease of both sides; large area of complete consolidation on the left, with dulness, bronchial breathing and voice, and no râles; surrounding this broncho-vesicular breathing, with many râles. Signs in the right lung similar to those previously present over the left. (*Holt.*)

.—The disease may stop at any one of these stages and resolution take place.

d of the process subsiding the symptoms persist and the con-
ed areas are noted to gradually extend until the greater portion

of both lungs posteriorly are involved. Over this area there is marked dulness, exaggerated bronchial breathing and bronchophony.

In the adult the physical signs are essentially the same as those found in the child.

In all forms of broncho-pneumonia and especially as it occurs in those of advanced years and those debilitated by some chronic disease, there may never be any signs pointing to consolidation of the lung tissue as the lesions may be small and widely scattered. Holt emphasizes the following points: (1) That where there is consolidation it is usually incomplete, because there are small areas of healthy lung tissue between the hepatized portions; (2) that the signs of consolidation usually shade off gradually; and (3) that both sides are almost invariably involved, although one side usually to a greater degree than the other.

Complications and Sequels.—Complications are not as frequently encountered in broncho-pneumonia as in the lobar form. Occasionally pericarditis and endocarditis occur but not often. Pleurisy with or without an effusion is not common. In children especially in those under bad hygienic conditions, various forms of stomatitis and gastro-enteritis are frequently seen. Purulent meningitis is relatively uncommon. Small multiple abscesses may occur especially in the aspiration and metastatic types of the disease. As a rule in the cases which recover resolution is complete but in some instances a diffuse fibrosis of the lungs takes place. This may be associated with dilatation of the bronchi.

Diagnosis.—Primary broncho-pneumonia in which the onset is sudden may be confused with the lobar form. It is to be borne in mind that broncho-pneumonia occurs almost exclusively in children under two years of age and that lobar pneumonia is unusual at this age. While the area of consolidation may simulate that found in lobar pneumonia a careful examination will usually show a smaller focus on the opposite side. In adults the distinction between secondary broncho-pneumonia and frank lobar pneumonia is usually easy. The onset, symptoms and physical signs are radically different. In atypical cases of lobar pneumonia, especially in the aged, mistakes are frequently made. In young infants confusion may arise as to whether we have to do with broncho-pneumonia or *congenital atelectasis.* If the latter is present the temperature is often subnormal and the cyanosis marked. Physical signs are scanty or absent. In a child previously healthy the occurrence of cough, fever, dyspnea and cyanosis points to broncho-pneumonia and in such cases the physical signs are usually distinctive.

The differentiation between *tuberculous* and *non-tuberculous broncho-pneumonia* is not easy. Confusion is most apt to occur in those cases in which the disease is prolonged beyond three or four weeks. As a rule the symptoms are more pronounced in the tuberculous form and the wasting and prostration are greater. While tuberculous broncho-pneumonia may involve any portion of the lungs the localization of signs at the apex or in the axilla are suggestive of this type as the non-tuberculous form shows a marked preference for the posterior and inferior portions of the lungs. If sputum can be obtained either from vomited material or by swabbing the back of the throat, the detection of tubercle bacilli at once determines the diagnosis. In children under two years of age a positive tuberculin reaction is extremely suggestive.

In cases with marked nervous symptoms the distinction between roncho-pneumonia and meningitis is often difficult. In such cases a umbar puncture should always be performed.

INFLUENZA

In his monograph on influenza Leichtenstern writes: "The winter 1889–90 is indelibly engraved on the history of great epidemics. n influenza epidemic greater than any before arose in the far East, read like a hurricane through Europe and thence over the greater art of the earth. Four decades after the last European pandemic 847–48) the medical profession of our day found itself confronted by a ew disease, which up to that time had been known to them only in e history of medicine. The interest which the affection everywhere icited, the competition which it kindled in all civilized lands to apply the new disease the progress and the acquisitions of modern medicine, lvanced our knowledge of influenza in every direction." This quotaon is a fitting introduction at the present time. Again the long interval hich elapsed between the pandemic of 1889–90 and that of 1918 und physicians more or less unfamiliar with the disease and stunned em with the appalling toll of death which it exacted.

In reviewing the history of previous pandemics the disease seems to ive varied greatly in severity. Rush, for instance, in writing of the idemic of 1789–90 in Philadelphia describes the affection as one characrized by "a perpetual coughing" but "with few exceptions the malady oved fatal only to old people and to persons weakened by pulmonary mplaints."

Walshe,[1] writing at a time midway between the pandemic of 1848 and at of 1889 states that—"Fortunately the virulence of the disease falls r short of its activity—two per cent. of those attacked being probably fair estimate of the average mortality. Influenza in truth, scarcely ever lls those it affects, unless, aged and debilitated, they have already one ot in the grave."

Whether the older physicians who had experience with the disease 1889–90, through the lapse of time, had lost their impression of the verity of influenza or not, the fact remains that but few of the present neration had any appreciation of its fatal effects. Most of us looked on the disease as one in which a large number of people were almost multaneously stricken but that few died. Stengel,[2] on the other hand, s pointed out from personal experience, that the epidemic of 1889–90 as characterized by a death-rate far in excess of the normal and that e increase in the mortality rate was due to severe pulmonary complicaons. These complications differed in no way from those encountered the recent epidemic, except, perhaps in the fact that they occurred ith greater frequency and therefore caused a higher death-rate.

Although the exciting cause of influenza is at present subject to me difference of opinion there is no question as to its being as distinct entity as any of the other general or acute specific diseases and that obeys the laws of this group. Because of the anatomical lesions produced the disease may properly be classed among those affecting t respiratory organs, though it is by no means limited to these.

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[1] Diseases of the Lungs, Fourth Edition, 1871.

[2] *Medical Clinics of North America*, Nov., 1918.

There can be no doubt that the term influenza or "grippe" is ve loosely used and that this designation when applied to catarrhal col is not strictly true. Influenza proper occurs in a wide-spread epiden or pandemic, lasts for a brief time and then ceases. Small resid epidemics may recur for a year or so following the main attack but th it practically disappears. Catarrhal colds, on the other hand, always quite constantly present during the winter months and at tin may occur as a mild epidemic. While they may be of the same nat as true influenza they lack many of the chief characteristics of the lat disease. As Leichtenstern has pointed out catarrhal colds have t same superficial resemblance to true influenza that cholera nost has to true cholera.

In regard to the recent epidemic, as it appeared in this count there is some doubt as to whether its onset should be placed early in fall of 1918 or whether the localized epidemics which appeared in ma of the army cantonments during the winter of 1917–18 may not considered the forerunners of the main attack. Certainly many of th local epidemics bear a striking resemblance to the disease which spr with such explosive suddenness in the autumn of 1918.

While influenza has, in general, fairly definite characteristics, must keep clearly in mind that it is subject to a number of variati both pathologically and clinically, and that furthermore it varies grea in its manifestations in different localities. Indeed the description the disease as seen by one observer in one locality often differs so m from that of another in another locality that it is hard to believe t we are dealing with the same disease. It is for these reasons t anything like a lucid description of the disease is a difficult task.

At the height of the epidemic the disease presents itself in vary degrees of severity which range from the simple, uncomplicated fo to fulminant cases of broncho-pneumonia and atypical lobar pneumo In addition a number of cases of typical lobar pneumonia are encounte

Etiology.—The incubation period is not definitely establish it has been estimated by different observers as being from a few ho to four days.

Weather conditions *per se* do not seem to play any part in the spr of the disease. Although the two last epidemics in this country h occurred in the autumn and early winter months undue emphasis sho not be placed on the fact that respiratory affections are more preval during this season or that public meeting places are less well ventila than in the warm months. The disease strikes with equal intensity subtropical and tropical regions. It is natural to expect, howe that the spread of the disease is favored by the housing of large numb of people in close quarters, such as occurs in barracks, or the gathe together of people in places of amusements, churches, schools, Under these circumstances the infecting agent can more readily transferred from individual to individual than would otherwise be case. It has long been noted that practically all of the great panden have originated in the East and have spread throughout the W The relatively rapid extension of the disease from place to place and sudden appearance in a locality some distance from an infected reg led the older epidemiologists to believe that the infecting agent was air born. It is now generally accepted that the disease is transferred from individual to individual and that its progress is that of the ordinary rate of travel.

Transmission of the disease occurs largely, if not entirely, through the inhalation of infected droplets which have been thrown into the air by coughing and sneezing and it is largely for this reason that the gathering together of people in crowded places is inadvisable and that the wearing of gauze masks should be practised by those caring for influenza patients. In both the epidemic of 1889–90 and that of 1918 it was noted that institutions shut off from communication with the outside world frequently escaped the infection. In the recent outbreak a tuberculosis sanatorium which I visit escaped the infection entirely during the height of the epidemic. Toward its close one of the nurses went home for a visit and returned to the institution with a mild attack of influenza. A small outbreak occurred among those brought in immediate contact with her and of the ten individuals who acquired the disease, one died.

Influenza attacks all ages. Those in the third and fourth decade of life, however, bear the brunt of the attack. Based on what we had read of the pandemic of 1889–90, many of us were of the belief that while the morbidity rate was extremely high, the mortality rate was relatively low and that only those of advanced years or those debilitated by a chronic illness were apt to die. It came as a distinct shock, therefore, to encounter the appalling death-rate in the recent epidemic and particularly among those in the prime of life. All classes seemed to be equally affected. There was one group, however, in which the disease struck with particular intensity, namely—pregnant women. In my own experience this was undoubtedly the most distressing feature of the epidemic as I saw it in the wards of one of the Emergency Hospitals of Philadelphia. Many of the women aborted and a very large proportion died. This is to be attributed partly to the severe toxemia of the disease and partly to the lack of prompt attention for premature labor and abortive cases. Woolston and Conley[1] report 101 cases of influenza-pneumonia complicated by pregnancy. Of this number, fifty-two (51.4 per cent.) died and of the forty-nine which survived, twenty-one (42.7 per cent.) aborted or went into labor prematurely. During the height of the epidemic the Pre-natal Clinic at the Phipps Institute had thirty-five pregnant women under observation. While the number is small, it would seem that the prompt attention that these women received had some bearing on the fact that but one died and two aborted.

At the present time it is impossible to estimate with anything like accuracy, either the morbidity or the mortality rates in this country. In the city of Philadelphia the number of cases reported from September 23d to November 8, 1918, was 48,131 which represents about 2.5 per cent. of the population. There is every reason for believing, however, that this number is far below those actually affected. The number of deaths attributed to influenza and pneumonia during this period was 12,687; the percentage of deaths to cases reported was 26.5. The highest number of deaths in any one day was 711, which is more than 100 above the average weekly death-rate from all causes in Philadelphia. In the country at large Soper[2] states that from the statistics available in the forty-five principal cities the death-rate was 3.24 per thousand. There were 127,584 deaths from all causes. Had there been no epidemic of influenza-pneumonia, it is estimated that there would have been 47,967 deaths. The difference, therefore, represents the deaths due to the pandemic, namely—79,687. If this rate prevailed throughout the continental United States, there would have been 341,021 deaths. To this must be added the 19,249 deaths occurring in the army camps, making a total of 360,450 deaths. While it is admitted that these figures represent little more than a guess, they serve to indicate the extent of the pandemic. Tice[1] in comparing the estimate of 20,000,000 deaths as the toll of the recent war in four and one-half years, believes that during an equal period of time, influenza at the epidemic rate would have caused 108,000,000 deaths!

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[1] *Jour. Am. Med. Assoc.*, Dec. 7, 1918.

[2] *Ibid.*

When the epidemic appears in a new locality it is apt to be preceded by a few scattered cases, the nature of which is not always recognized. The disease then usually spreads with explosive suddenness, reaches its maximum effect in about ten days and in from three to four weeks the epidemic has spent its force, although it may persist in a much less aggravated form three or four weeks longer. Small localized epidemics may recur for several months after the subsidence of the main epidemic and during the two or three years following there may be small local outbreaks in those places where the disease had been prevalent previously.

Bacteriology.—In 1892 Pfeiffer described a small gram-negative bacillus which he believed to be the specific cause of influenza. The acceptance of this organism as the causative factor of the disease gained almost universal acceptance although no one had the opportunity of verifying its relationship to epidemic influenza. When the recent epidemic appeared bacteriologists immediately sought for the influenza bacillus. Almost at once, however, it became apparent that either the influenza bacillus was not always present or that many workers were unable to isolate it. In going over the reports which have appeared during the past few months one has the greatest difficulty in forming any definite opinion as to what the actual rôle of the *bacillus influenzæ* is. The question is further confused by reason of the fact that one observer, for example, who in the beginning of the epidemic believed Pfeiffer's bacillus to be the sole cause of the disease later had doubts as to whether it plays any part whatever; and again one who originally thought it had no relationship to epidemic influenza reversed his judgment in favor of the organism being the only cause of the disease. Out of this confusion one is able to separate three opinions: (1) That the bacillus influenzæ is the specific cause of the disease; (2) that it plays little or no part as a causative factor; and (3) that the bacillus opens the door, so to speak, to various other organisms, such as the pneumococci, streptococci, staphylococci, Friedländer's bacilli, etc., and that these last-named germs are often the causes of the various complications. The last-mentioned conception offers a satisfactory explanation for the extreme variations which have occurred not only as to severity but also as to protean aspects of the disease. It is to be borne in mind, however, that there is already sufficient evidence to sustain the belief that mixed infection is not necessarily essential to the occurrence of pneumonia.

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[1] *International Clinics*, vol. i, Twenty-ninth Series, 1919.

Morbid Anatomy.—The pathological changes which are encountered in influenza are difficult to describe because of the complications, combinations and variations arising from differences in the stages of the disease at which the patient died.

In order to obtain a clear conception of this infection, it is necessary to keep in mind that there is more than one organism at work in practically all cases in which serious pulmonary complications occur. In a number of cases which have been carefully studied there appears to be a fairly definite sequence of events: (1) A primary infection of the upper respiratory tract and the bronchi by the influenza bacillus; (2) a secondary invasion of the inflamed bronchi by pneumococci (commonly type IV), which enter the lung and produce either broncho- or lobar-pneumonia and (3) a tertiary invasion by streptococci (hemolytic or non-hemolytic) which infect the pneumonic areas.—(Opie, Freeman, Blake, Small and Rivers, Camp Pike Pneumonia Commission.[1]) In some instances the

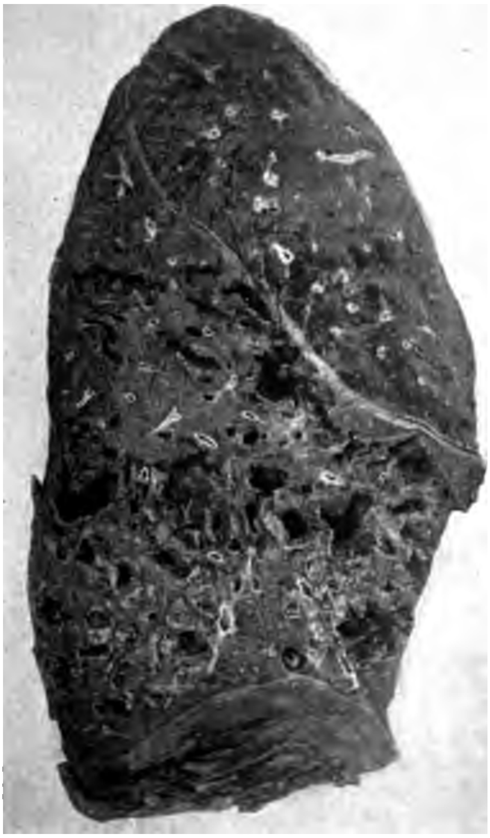

Fig. 283.—Extreme dilatation of bronchi and softening of the pulmonary tissues. (*Camp Pike Pneumonia Commission.*)

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[1] *Jour. Am. Med. Assoc.*, Feb. 22, 1919.

pneumococci play no part, the streptococci being the secondary invaders. In other instances the secondary invaders may be the staphylococcus or Friedländer's bacillus, or the influenza bacillus itself may extend into the pulmonary tissues and give rise to pneumonia.

The initial infection with the *bacillus influenzæ* causes a more or less intense inflammation of the mucous membrane of the upper respiratory tract, the trachea and the bronchi (see Fig. 219). The pharynx and tonsils appear red and inflamed. The nasal mucosa is swollen and injected and on laryngoscopic examination the larynx is frequently see

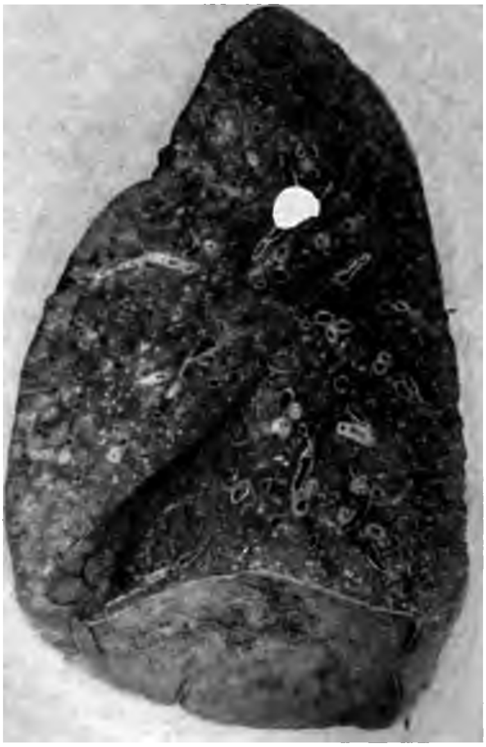


Fig. 284.—Broncho-pneumonia. (*Camp Pike Pneumonia Commission.*)

to be deeply injected as is also the mucosa of the trachea. In some instances early extension of the inflammation occurs to the frontal and ethmoid sinuses and the antrum of Highmore. Usually the process extends downward and involves practically all of the bronchi. The distribution is not necessarily uniform, the upper air tract may be but slightly involved, the brunt of the infection occurring in the bronchi. In other cases the bronchi seem to be but slightly involved while the upper air tract and the trachea are intensely inflamed. In still other instances a limited portion of the bronchial tree in one lung is much more involved than the remainder. In a considerable number of cases a purulent bronchitis and bronchiolitis develops. When these conditions exist the lungs are voluminous and show but little tendency to collapse even on section. If purulent bronchitis is present the muco-purulent material wells out of the cut bronchi. In twenty-seven out of thirty cases of this type Opie and his co-workers recovered the influenza bacillus.

The mucous membrane of the bronchi is not only intensely inflamed, but in addition the influenza bacillus penetrates the wall producing varying degrees of softening. This may extend to the surrounding pulmonary tissue which also becomes softened and in extreme instances becomes of a pulp-like consistency. The softening of the bronchial wall frequently leads to the acute dilatation of the bronchi which in extreme cases gives the lung a honeycombed appearance (see Fig. 283). It has long been recognized that the most constant factor in chronic

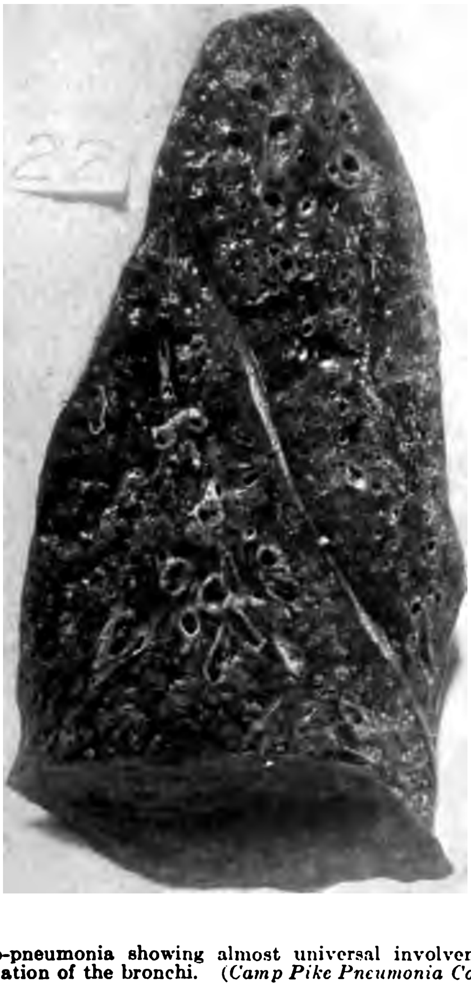


Fig. 285.—Broncho-pneumonia showing almost universal involvement of entire lung. Also dilatation of the bronchi. (*Camp Pike Pneumonia Commission.*)

bronchiectasis is the influenza bacillus. It is probable, therefore, that even in mild cases in which there is no noticeable change in the caliber of the bronchi that the weakening of the wall and the persistence of the influenza bacilli eventually lead, in the course of months or years, to a definite bronchiectatic condition.

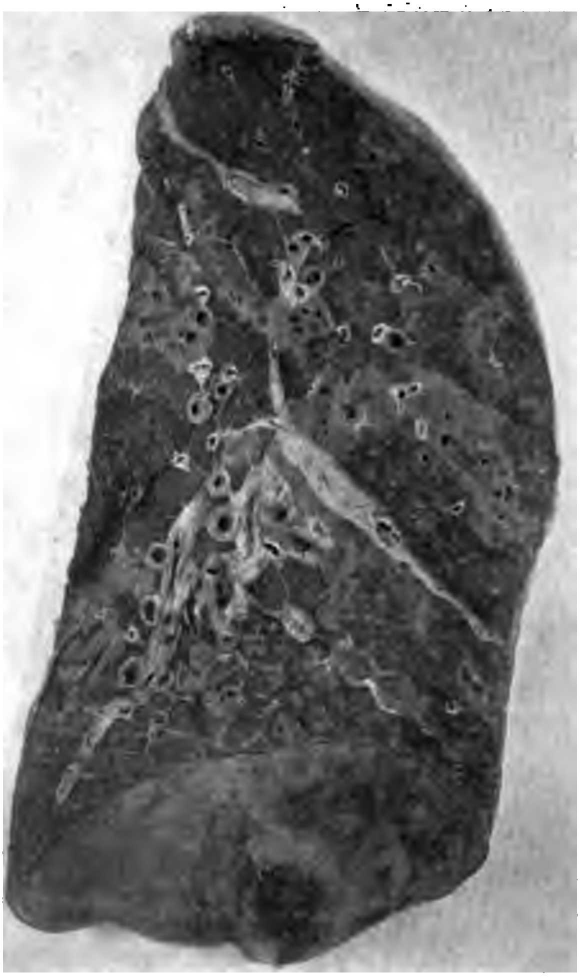
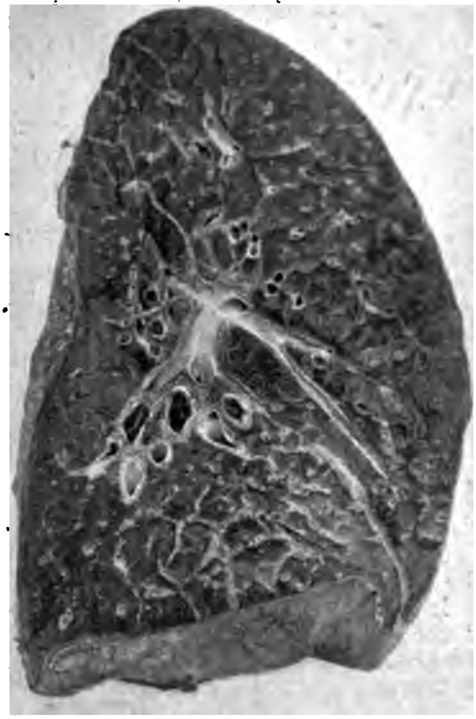


FIG. 286.—Broncho-pneumonia showing large confluent areas of consolidation. (*Camp Pike Pneumonia Commission.*)

This primary infection of the bronchi creates what Allen J. Smith has aptly termed "an open wound" which is easily infected by the secondary invaders. Furthermore the disease is characterized by a marked lowering of resistance to material infection.

The most common lesion in the lungs is a ***broncho-pneumonia.*** The broncho-pneumonia may be caused by the influenza bacillus alone by rect extension from the bronchi. More often, however, the pneumoccus is responsible for the broncho-pneumonia. The broncho-pneumonic eas are often limited to or most marked about the hilus of the lung. me observers have emphasized the predilection of the lesions for the riphery of the lungs and have also directed attention to the hemoragic character of the process. In many cases there is relatively little nsolidation. The pneumonic areas may consist of small shot-like dules or smaller areas may become confluent forming areas the size an English walnut or larger (see Figs. 284, 285, 286). The color y be blackish-red, red or grayish-red. Even in cases in which constion is not a marked feature and the amount of consolidated tissue



IG. 287.—Interstitial type of broncho-pneumonia. (*Camp Pike Pneumonia Commission.*)

relatively slight, there is often a thin, copious, bloody exudate in the onchioles and lung tissue which pours out of the nostrils after death.

In a certain number of cases the interstitial tissues are involved nstituting what has been termed an *interstitial broncho-pneumonia* e Fig. 287). In these cases microscopic examination shows thickening the walls of the bronchi and bronchioles and the alveolar walls in the mediate neighborhood of the bronchioles are also thickened by an ltration of mononuclear cells. At one stage the peribronchial inration presents a gross appearance closely resembling tubercles, l MacCallum suggests that it is possible that these lesions have perhaps been mistaken for tuberculosis when the condition follows measles. At a later stage the interlobular septa become greatly thickened and stand out conspicuously as whitish-yellow lines, marking out the whole lobulation in polygonal fields. In each of these fields there may be three or four projecting nodules which represent the thickened bronchi.

One of the chief characteristics of influenza is the occurrence of an *atypical pneumonia* which has some of the features of both broncho- and lobar pneumonia. The current opinion at the present time is that these so-called pseudo lobar pneumonias are primarily lobular and that the gradual merging of a number of isolated patches of consolidation may involve a large part or even all of one lobe. This condition is often brought about by the streptococcus hemolyticus. The gross appearance of this form of lobar pneumonia is not that of the true type of the disease for instead of being red or grey in color the lesion presents a variety of tints of yellow varying from a pale yellow to a reddish yellow. These various colored areas appear to be different stages of a broncho-pneumonic process. Furthermore the cut surface of the atypical form lacks the granular feel of true croupous pneumonia and is also much moister. This type of pneumonia was very prevalent in the army camps during the winter of 1917–18 and seemed in most instances to be caused by the streptococcus hemolyticus. A complicating empyema was frequently present (see Streptococcic Empyema).

A *hemorrhagic form of broncho-pneumonia* in which infection with the streptococcus hemolyticus occurs shortly after or coincidently with the primary influenza infection was quite common. In these cases the lungs are deeply congested, and on section a copious, bloody fluid exudes. In addition, there may be areas of hemorrhagic infiltration. The lower lobes are often enlarged, heavy and often almost completely consolidated. On section the involved portion of the lung presents a bluish or bluish-black color. The lung substance is less friable than in croupous pneumonia and the cut surface is soft and smooth. When the upper lobes are involved the process is less uniform in appearance. Some portions are bluish in color and of a firm consistency; others are red and less firm while the extreme edges of the lung are emphysematous (Symmers). The condition is not unlike that seen in the pneumonic form of plague. It has also been described as a hemorrhagic pneumonitis. In these cases there is every evidence of a profound septicemia which clinically produces symptoms similar to those seen in severe puerperal fever. The infecting organism can be recovered from the heart's blood and lungs.

Still another variation, caused by the streptococcus hemolyticus, is what has been termed the *relaxed form of pneumonia* (see Fig. 288). In these cases the chief characteristic is the shrunken, flabby appearance of the lung. On section it presents a deep red and congested appearance and not infrequently there is abscess formation. The latter is often situated just beneath the pleura and may rupture into the pleural cavity producing an empyema.

In addition to these atypical forms of pneumonia many cases of true lobar pneumonia are encountered which may be primary or occur as a sequel to an attack of influenza.

Edema.—In some fulimant cases edema of the lungs occurs early. In other instances, the edema occurs relatively late when the right heart begins to fail.

Acute vesicular emphysema is quite constantly found in association with broncho-pneumonia. These emphysematous areas are apt to be marked along the free margin of the lungs and not infrequently rupture. Instances of rupture near the root of the lung with extravasation of air into the mediastinal tissues have been noted. A number of cases of *subcutaneous emphysema* appearing first in the tissues of the neck, have been reported. This may result from the rupture of an emphysematous bleb at the apex, although it has not been possible at autopsy to demonstrate the point of rupture. In one of the cantonments Torrey and Grosh[1] state that the predominant lesion was a generalized acute emphysema. The lungs were gray in color at the apex and deep red at the base. The surface of the lungs was glistening, showed the markings of the ribs and emphysematous bullæ. The lungs were in a state

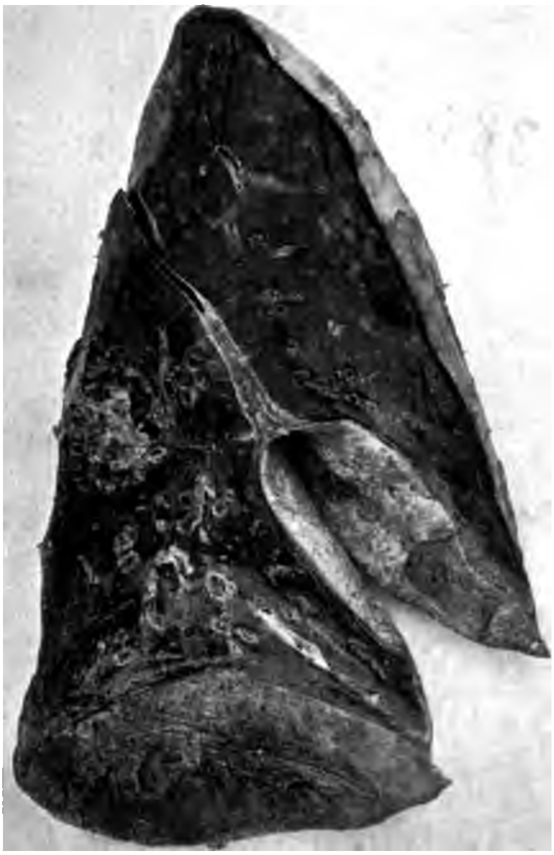


Fig. 288.—Note relaxed condition of lower lobe. Abscess beneath the visceral layer of pleura. (*Camp Pike Pneumonia Commission.*)

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[1] *Am. Jour. Med. Scs.*, Feb., 1919.

of inspiratory distention, did not collapse and were tough at the apex and very friable at the base. On section bloody serum exuded; the bronchi stood out prominently and they contained a thick creamy pus. Scattered through both lungs were numerous small nodules varying in size from a millet seed to a bean. There was also an intense bronchitis and peribronchitis and marked venous stasis. The latter condition was probably due to the extreme distention of the lungs and clinically gave rise to the extreme lethargy, cyanosis, dyspnea and bloody sputum.

Abscess formation is not infrequent. The abscess may be (1) localized, having its origin within a patch of pneumonic consolidation; (2) it may occur in the interstitial tissues either in association with broncho- or lobar pneumonia or it may arise by absorption from an empyema; (3) there may be small multiple abscesses clustered about the bronchi.

The *pleura* is frequently the site of a fibrinous exudate. Effusions are relatively common. They may develop coincidently with the pneumonic process but more often occur as sequels. In the beginning the effusion is often hemorrhagic in character but it quickly becomes purulent. The effusion may be free or encysted. The number of loculated effusions during the past epidemic was unusually large and were always to be suspected when the physical signs and fever persisted. Among the 263 cases seen in the University Hospital a pleural effusion was noted in 26 (10 per cent.). In this connection the article on streptococcic empyema should be read.

The *pericardium* is not involved as frequently as one would expect. Effusions are not common and in spite of the complicating pneumonia the dry or fibrinous form does not often occur. In fatal cases, which have been characterized by marked septicemia, changes in the myocardium are usually noted.

In a very large proportion of cases the mucous membrane of the *intestines* shows the presence of small hemorrhagic areas. Hemorrhage into the adrenals also occurs in a few instances.

In a number of cases *hyaline degeneration of the rectus abdominis muscles* has been noted. The muscles may be completely ruptured, probably as the result of coughing which causes a tear at the weakened point. A large extravasation of blood sometimes occurs into these muscles and in some instances the streptococcus hemolyticus has been recovered from the affected area.

There are no noteworthy changes in the other viscera aside from those encountered in febrile conditions.

Involvement of the *ear* and *sinuses* may occur as a complication or sequel of the disease.

Meningitis may be encountered in the severer forms of influenza. Lamb[1] reports its occurrence in 7 cases out of 624 influenza pneumonias. The infecting organism was the pneumococcus in 6 instances; the streptococcus hemolyticus was obtained in the remaining case. Neal[2] has observed 3 cases caused by the influenza bacillus.

A late sequel of the disease is *encephalitis*. The encephalitic area in its typical form consists of innumerable dense aggregations of "flea-bite-like" dots of blood, between which the tissue is softened and of a gray or grayish-red color (Leichtenstern). Secondary hemorrhages may occur in the softened area. The foci are usually sharply defined, nearly situated in the gray matter of the cortex and central ganglia and ;tically always symmetrically located in both cerebral hemispheres. e instances there are patches of congestion or even meningitis. clei of the motor cranial nerves, especially the third nerve, are ·quently involved.

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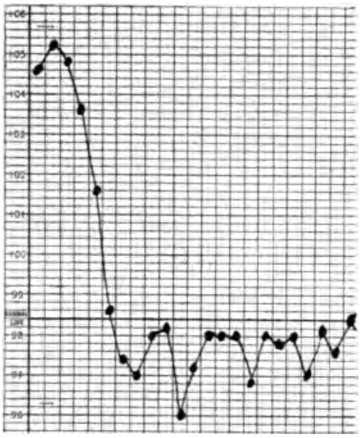
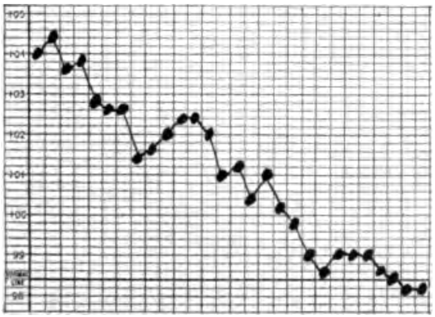
[1] *Jour. Am. Med. Assoc.*, April 19, 1919. [2] Ibid., March 8, 1919.



.—Simple influenza. Fever of short duration, with sharp decline. (*Medical Clinics of North America.*)

ıptoms.—The disease, as a rule, begins abruptly with malaise drowsy feeling. Distinct chills are not common but chilliness is ıtly complained of. Pain is one of the characteristic features. :en quite generally felt all over the body but is apt to be especially n the legs, head and eye-balls, and across the small of the back, the



290.—Simple influenza. Fever of four days' duration. Gradual decline. A few both bases. No other physical signs. (*Medical Clinics of North America.*)

times being as severe as in smallpox. In addition, in the respirarm, there is commonly coryza, sore throat, soreness or actual pain :hest, and cough, which at first is apt to be unproductive and later ›anied with mucopurulent sputum. In not a few cases a marked ıt bronchitis develops in the later stages of the attack. The sputum, in addition, may be blood-tinged and at times consists of almost pure blood. Severe epistaxis occurs in a few cases. Gastro-intestinal disturbances are relatively common for a day or two, but only in exceptional instances can they be looked upon as severe enough to consider the case one of the true gastro-intestinal type. Many patients complain of nausea and vomiting for the first few days of the attack. Vomiting of blood and a hemorrhagic diarrhea are encountered in a few cases.

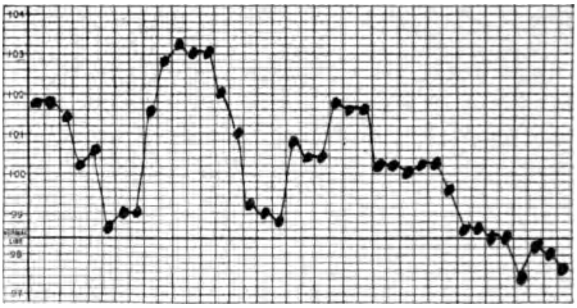


Fig. 291.—Simple influenza. Eight days' duration. Marked congestion of pharynx and tonsils. Gastro-intestinal symptoms for two days. A few scattered râles at bases of lungs. (*Medical Clinics of North America.*)

Jaundice is not often present. Even in cases which are dominantly of the respiratory type nervous symptoms may be pronounced owing to the severe toxemia. Insomnia, occasionally intractable in character, is often complained of and a low muttering or active delirium may be present.

In previous epidemics mention is made of a very mild type of the disease in which there is little or no fever; none of this type was seen by me in the recent outbreak. Fever is practically always present and at the onset may be quite high. The temperature chart may show a high continuous type of fever for a few days, or it may rapidly fall to normal in the course of thirty-six to forty-eight hours (see Fig. 289). More often it gradually falls by lysis (Fig. 290). Often the temperature falls irregularly, and at times the fever curve may be marked by sharp remissions (Fig. 291). Not uncommonly when the fever subsides the temperature may become subnormal and may continue this way for days and often

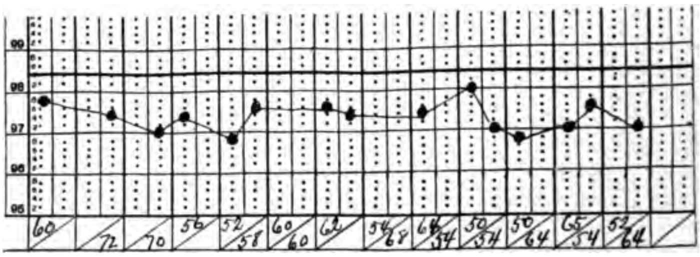


Fig. 292.—Subnormal temperature and low pulse-rate during convalescence from influenza. (*Medical Clinics of North America.*)

for weeks (Fig. 292). In a few instances I have seen what was apparently a relapse of the influenza infection without serious implication of the lung itself. In such cases after a febrile period of three or four days the temperature again rises for a brief period (Fig. 293). If the second febrile disturbance is prolonged, however, it is more than likely that the secondary rise has resulted from a broncho-pneumonia or a small collection of fluid.

An interesting feature of the disease is the slowness of the pulse compared to the febrile disturbance. Even with a complicating pneumonia, the pulse-rate is often unduly slow. During convalescence a marked bradycardia (40 to 50) is relatively common. A study of these cases with the electrocardiograph showed nothing abnormal other than the wide spacing between beats.

Cyanosis is not present in simple uncomplicated influenza. It is very frequently present, however, in cases with severe pulmonary complications and is usually indicative of severe toxemia rather than circulatory failure. The blood pressure readings are extremely variable. They give but little indication of the severity of the disease or the state of the circulation.

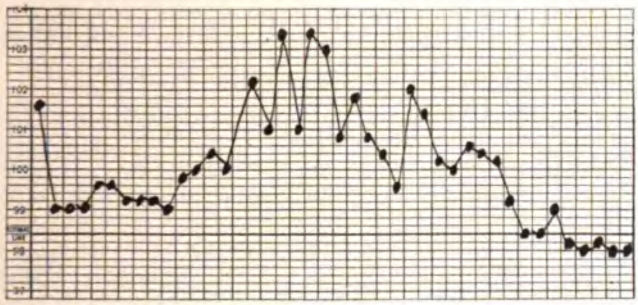


FIG. 293.—Relapse. Patient taken ill two days before admission. Temperature declined to nearly normal, remained so for two days, and again rose. A few râles at right base. (*Medical Clinics of North America.*)

The respiratory rate even in uncomplicated cases is subject to marked variations. It may be quite high, but rarely continues so unless there is a complication—lobar or broncho-pneumonia. Marked irregularity in the curve of the respiratory rate is not infrequent.

The urine commonly shows the presence of some albumen and a few casts but no more frequently than ordinarily occurs in other acute infections. Severe nephritis is occasionally encountered. Free diuresis was noted in most of the cases but this may be attributed to the fact that an abundant water intake was encouraged. Retention of urine and an over-distended bladder was met with sufficiently often to put one on one's guard in treating these cases.

se sweating is not uncommon. Herpes is not common even es with a distinct lobar consolidation. *Subcutaneous emphysema* reported as occurring in a number of cases, usually those with ting pneumonia. I saw an example of this in a child in which ema involved one side of the neck, and all of the anterior aspect and abdomen. The condition apparently is not serious rs in a few days. Its causation is not clear.

A leukopenia or normal white-cell count is the rule in simple influenza. A leukocytosis is always suggestive of a complication.

The following white cell counts were obtained in 54 cases in the University Hospital.

	Uncomplicated cases	Pneumonic cases
15,000+	3	12
9000 to 15,000	13	21
2 to 9,000	38	26

These figures are in accordance with those reported by a number of observers. With improvement a leukopenia is often changed into a leukocytosis. It has been said that in broncho-pneumonia the leukocyte count is not usually over 15,000 and that counts higher than this point to the lobar type of pneumonia.

Physical Signs.—In simple influenza of the respiratory type the physical examination indicates the presence of a bronchitis. This is nearly always bilateral but may be more marked on one side than the other. The râles may be fairly generally distributed over both lungs but commonly they are limited to the bases of the lungs posteriorly. Occasionally the bronchitis involves only a portion of the bronchial tube in one lung. The râles may be subcrepitant in character but commonly large, mucous râles are also present.

As the morbid process in simple influenza is confined to the bronchi and peribronchial tissues, there are no areas of consolidation. The heart sounds are relatively slow even when considerable fever is present; during convalescence the pulse-rate may drop down as low as 40 beats per minute. Aside from this slowness of the pulse-rate the heart rarely shows anything abnormal.

Influenza Broncho-Pneumonia.—Whether broncho-pneumonia is to be looked upon as a complication or as an integral part of the disease is not clear. As has been pointed out in the section on morbid anatomy the inflammation of the bronchial mucous membrane constitutes an open wound which is readily infected with other organisms, notably the pneumococci, streptococci, staphylococci, Friedländer's bacillus, etc. Furthermore there may be two or even more of these secondary infections. Simple uncomplicated influenza is rarely fatal. In the vast majority of the fatal cases the disease has extended beyond the bronchi and involved the parenchyma of the lung. Ascribing death to influenza has been due in many cases to the fact that the physical signs are often so indefinite that there is no suggestion of pulmonary consolidation.

The duration of simple influenza is rarely longer than six or seven days and in addition the evidences of toxemia are wanting. Therefore, if the fever persists beyond this time and if there are also marked toxic symptoms a broncho-pneumonia is to be suspected.

The onset of the broncho-pneumonia varies. It may develop gradually and become apparent five or six days after the onset of the influenza. In other instances it is fulminant in type and occurs in a day or so from the onset of the influenza or may appear almost simultaneously. The slowly developing type is usually due to a pneumococcus infection while the fulminant cases are commonly due to the streptococcus hemolyticus, less frequently one of the staphylococci. Evidences of a severe septicemia such as occurs in puerperal sepsis, characterizes the fulminant cases. The distinction as to the relation between the infecting organism and the of the broncho-pneumonia is not absolute, as serious and rapidly ses may be pneumococcic in origin and relatively mild cases may to the streptococcus hemo- or staphylococcus.

he mild type of the disease the picture is not essentially t from that seen in ordinary -pneumonia except perhaps in tively slow pulse. The onset mild cases is usually identical at of simple influenza. In stances the fever declines and as though the disease would te in a few days. In some in- the temperature becomes or nearly so for a day or so and es again. This usually coin- ith the secondary invasion or ension of the influenza infec- the air cells and the develop- f a broncho-pneumonia (see 4, 295).

general it may be said that ptoms which characterize the m of influenza are greatly in- l when broncho-pneumonia de- and that in addition the res- symptoms and evidence of are often pronounced.

e fulminant type of the disease er, marked cyanosis, rapid and breathing are prominent symp- Marked stupor or even uncon- ss is common. In addition ient is plainly septic. The y have an icteroid tinge or distinctly jaundiced. In these characteristic lesion is usually se hemorrhagic pneumonitis ears a strong resemblance to of broncho-pneumonia seen pneumonic form of bubonic Associated with these cases cho-pneumonia there is nearly some acute emphysema just e case of ordinary broncho- nia. In some instances, how- e acute emphysema has be- neralized over both lungs and tes the dominant feature of ase. Clinically it is characterized by extreme lethargy, cyanosis, ctoration of bloody sputum, epistaxis, irregular temperature and se.

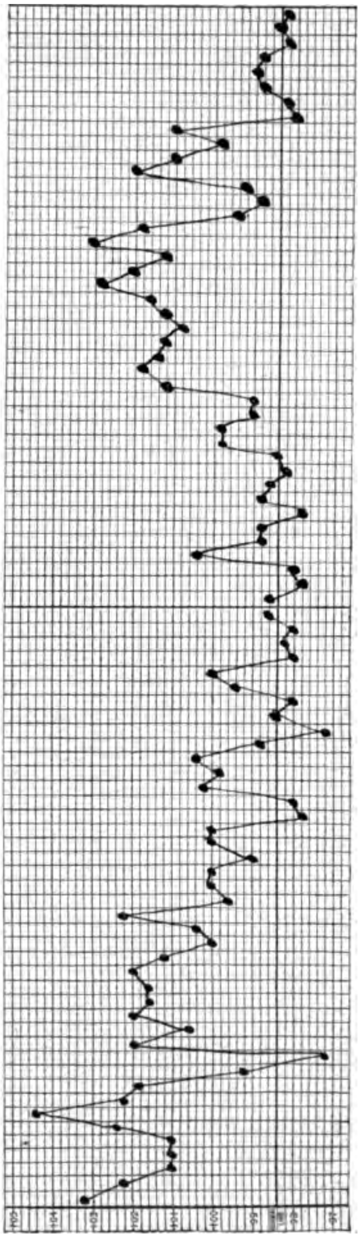


FIG. 294.—Broncho-pneumonia. (*Medical Clinics of North America.*)

Physical Findings.—Examination of the chest often fails to reveal any very striking physical signs. There may be no alteration of the tactile fremitus, no appreciable change in the percussion note and an absence of bronchial breathing. In other words the usual signs of pulmonary consolidation are wanting. As a result of post-mortem and X-ray examinations, we have come to learn that the broncho-pneumonic areas start in the great majority of cases about the hilus and that the area about the angles of the scapulæ should be carefully examined. While a diffuse bilateral bronchitis is almost invariably present in these cases, it will be noted that the râles are, in certain areas, notably near the angle of the scapula, of a sticky, resonating character. Râles of this nature are almost certain evidence of underlying consolidation. In addition it has been found that while the breath sounds may not be appreciably altered or obscured by râles, that the spoken voice is often exaggerated over the area in which the consonating râles are heard. In some instances the whispered voice is more distinctly transmitted than the spoken voice. Adams and Montgomery[1] ascribe this to the fact that the consolidated area is small and that the sound of the spoken voice is diffused by the surrounding lung tissue while no such effect occurs in the case of the whispered voice.

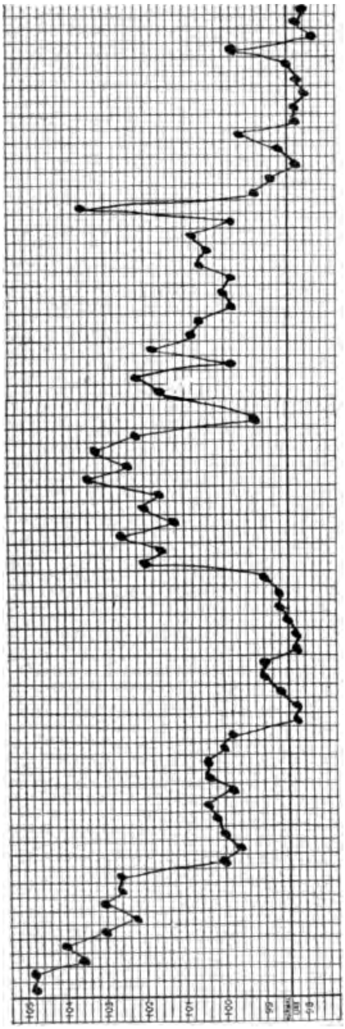


Fig. 295.—Broncho-pneumonia. (*Medical Clinics of North America.*)

The signs above described may never become any more marked. In many instances, however, as the infiltrated area increases, the physical signs become more and more marked, and eventually all of the signs commonly associated with pulmonary consolidation are obtained.

I have had the opportunity of studying many of these cases with Pancoast in the X-ray department of the University of Pennsylvania. This experience has convinced me of the importance of the signs referred to and has also demonstrated the fact that the broncho-pneumonic process is almost invariably far more extensive than the physical signs indicated.

When the condition is associated with marked general emphysema of both lungs the chest is in a fixed inspiratory position, the breathing is shallow and the veins over the upper chest and the neck are distended. The percussion note is hyperresonant over the sternum and vertebræ and he area of cardiac dulness is obliterated. There is a diffuse bronchitis nd in some areas the breath sounds are absent. Forced breathing may ring out râles in these silent areas.

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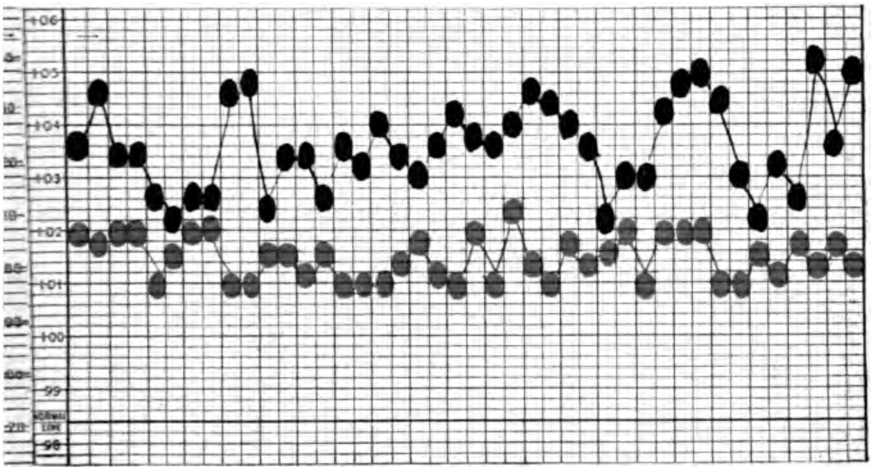
[1] *Jour. Am. Med. Assoc.*, April 5, 1919.

Influenza Lobar Pneumonia.—This type of pneumonia rarely begins ith an initial chill since, as a rule, it gradually develops from the fluenza. The typical tenacious rusty sputum is not often seen, uco-purulent or purulent sputum taking its place. The sputum may, owever, be blood streaked and at times consist of almost pure blood. erpes rarely occurs. The respiratory rate varies; it may be quite gh but is more often not especially accelerated. The fever course ay show marked remissions and even intermissions while the pulse-rate apt to be disproportionately slow in comparison to the fever. This is own in Fig. 296 which should be contrasted with Fig. 297, from a ase of true lobar or croupous pneumonia. Sero-purulent, purulent or emorrhagic effusions are apt to occur during the height of the disease in hese atypical forms rather than as a sequel as in the case of lobar neumonia.



296.—From a case of influenza pneumonia. Black—temperature. Red—pulse-rate.

The temperature, as a rule, falls by lysis.

Physical Signs.—The physical signs in these atypical forms of pneumonia are often identical with those encountered in the ordinary lobar neumonia. They may indicate the involvement of an entire lobe but ot infrequently the signs of consolidation (bronchial breathing, bronchophony, whispering pectoriloquy) are limited to an area almost the size of he palm of the hand near the angle of the scapula or in the axillary region. ver the rest of the lobe the percussion note is dull but there are commonly an absence of or diminished intensity of the breath and voice ounds.

Complications and Sequels.—*Pleural Effusions.*—One of the most requent of the after effects of influenza is a pleural effusion. In a few nstances the effusion develops almost coincidently with the pneumonia; n other instances, it occurs late in the disease or develops some days after convalescence apparently has been established. It is remarkable how frequently these effusions are mistaken for unresolved pneumonias. During the recent epidemic I saw in the course of two weeks no less than 6 cases of this sort and in every instance a purulent effusion was present.

When the effusion develops during the course of the pneumonia its detection is not always easy as fever, an accelerated respiratory rate and signs of consolidation are already present. Furthermore an effusion which develops in association with a consolidated lung is characterized by loud bronchial breathing and exaggeration of the voice sounds instead of the usual silence over collections of fluid. Errors can be avoided if the position of the apex beat of the heart is noted. If the apex is displaced away from the affected side and if in addition there is a nasal quality to the voice sounds an effusion should be suspected and a needle introduced. These effusions are not infrequently hemorrhagic in the beginning but they quickly become purulent and as a rule had best be treated surgically. (See also Streptococcic Empyema.)

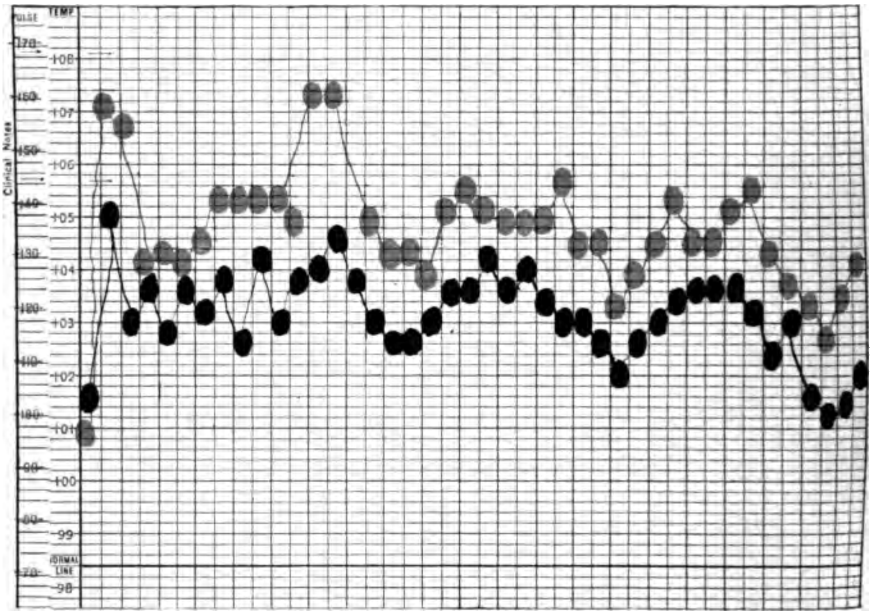


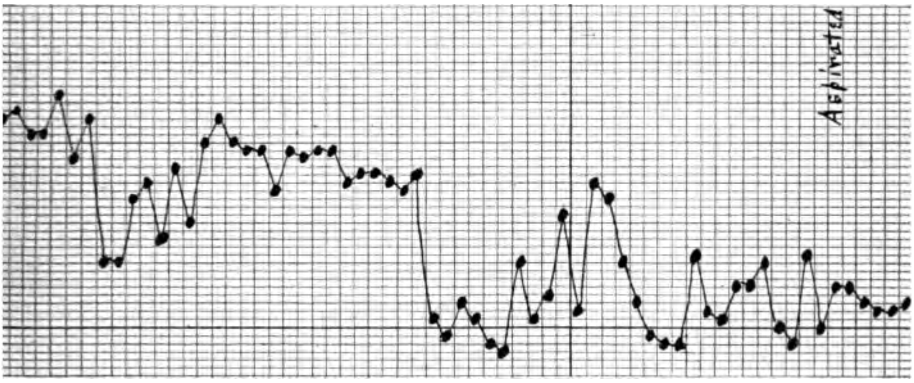
FIG. 297.—From a case of lobar pneumonia. Black—temperature. Red—pulse-rate.

If the temperature falls to normal for a few days and again rises or if instead of falling to normal it begins to show marked remissions or intermissions and the physical signs do not clear up an effusion should be suspected (see Fig. 298). A needle should be used and if this fails the patient should be X-rayed, if possible.

When the effusion is free in the pleural sac, its detection is not a difficult matter. Encysted effusions, on the other hand, are often extremely difficult to locate. The continuance of the fever and the indefinite haracter of the physical signs point strongly to such an occurrence but uite as often as not the location of the encysted fluid escapes detection ven when a needle is repeatedly introduced. This may be due to the eedle being too short, to the fact that the purulent material is too thick, r what is probably more often the case the physical signs fail to locate he pus sufficiently accurately. It is in this type of case that the X-rays re invaluable and without their aid it would be impossible to be certain ither as to the presence of or the location of many of these effusions.

In the recent epidemic the number of encysted effusions was notably igh. Of 35 empyemas operated on by Deaver in the University and ankenau Hospitals, 24 were localized, 10 were diffuse and one was n empyema necessitatis. Of the 24 which were localized, 13 were nterlobar.

In some instances the empyema has been caused by the rupture of a ulmonary abscess into the pleural cavity. Such an accident may be uspected, if during convalescence the patient is seized with a sudden, harp pain in the chest and in a few hours becomes critically ill. The emperature rises sharply, the pulse becomes rapid and small and the reathing shallow and very painful. The pain is usually located in the ower chest and upper abdomen. The abdomen is apt to be distended nd markedly tympanitic. The presence of fluid may be made out t the base of the affected side.



IG. 298.—Effusion following lobar pneumonia. Often mistaken for "unresolved pneumonia." (*Medical Clinics of North America.*)

Bronchiectasis.—Dilatation of the bronchi may occur as an acute rocess during the course of the primary infection (see Fig. 283). It is ncountered more often as a remote sequel, the symptoms and signs f the condition becoming manifest several years after the influenza ttack. The condition arises as a result of the bronchitis and peri-ronchitis produced by the influenza bacillus. The bronchial wall beomes softened and readily distended. As already pointed out this oftening may be so marked as to lead to dilatation of the bronchial ubes during the course of the primary attack. More often the dilataion occurs gradually and several years later presents the characteristic eatures of bronchiectasis.

Abscess Formation.—The formation of a distinct pulmonary abscess following influenza pneumonia is not very common in contrast to the frequent occurrence of interlobar collection of pus. The condition is to be thought of in every case in which there is a persistence of the symptoms and physical signs long after the pneumonic process should have cleared up. In a few instances the abscess has ruptured through the pleura and produced an empyema.

Tuberculosis.—There can be but little doubt that the recent epidemic will increase the number of cases of active pulmonary tuberculosis. Already a number of cases have come to light dating the onset of their trouble from an attack of influenza. It is to be borne in mind, however, that the persistence of a cough and physical signs following an attack of influenza does not necessarily mean that tuberculosis is the cause. Persistent and slowly resolving patches of influenza pneumonia, especially if located at the apex of the lung, may be mistaken for tuberculosis. In addition known cases of tuberculosis which have undergone an attack of influenza are going to offer considerable difficulty in determining whether the increase in the physical signs is due to an extension of the tuberculous process, or whether it is a slowly resolving influenza pneumonia which will gradually clear up. A conservative attitude must be maintained in respect to this latter group. A lung which has been infected with tuberculosis, even if the process has been arrested for some time, is extremely susceptible to acute respiratory infections, and because of the fibrous tissue and other changes about the lesion such infections are apt to persist for a much longer time than in a normal healthy lung. In the cases which have come to my notice it has not been possible to state at once whether the process was due to a lighting up of the old tuberculous lesion or whether the râles were due to the influenza attack and would eventually disappear, or, if râles had been present previously, that they would diminish. One should withhold an immediate opinion in these cases. The patient should be seen at intervals and by the end of six weeks or so, it should be possible to determine whether there has been a lighting up of the tuberculous process or not.

In regard to cases in which the tuberculosis apparently has been brought to light by the influenza attack, one should also adopt a conservative attitude. When the abnormal physical signs are located in the bases of the lungs there should be no confusion. When, however, fine crackling râles are heard in the apices the condition may or may not be tuberculous. In such cases the sputum should be repeatedly examined and in addition there should be careful temperature observations. If the lesion is due to influenza, the patient tends to improve, while if due to tuberculosis, he gradually becomes worse.

Upper Respiratory Tract.—As a result of the inflammation of the upper air tract involvement of contiguous structures may occur. Thus there may be involvement of the accessory sinuses, the middle ear or the mastoid. In common with other complications sinusitis, otitis media and mastoiditis were relatively frequent in some localities, and only occasionally encountered in others. Among the cases seen in the University Hospital these complications were not common.

Circulatory System.—The most characteristic change in respect to the circulatory system is the bradycardia. This condition is encountered in influenza more often than in any of the acute infectious diseases. The bradycardia may be absolute, that is the pulse-rate may range between

40 and 50 or it may be relative, 80 to 90 with a temperature of 103 or 104°F. The condition is apparently one of the neurotoxic manifestations of the disease. Electrocardiograph studies show no abnormalities.

Although there may be some slight increase in the amount of pericardial fluid inflammatory changes in the pericardium are not frequent. Involvement of the endocardium is rare. Individuals with a damaged heart muscle or valves, however, often bear the infection badly.

As a part of post-influenzal neurasthenia symptoms referable to the heart are not uncommon. These may consist of attacks of tachycardia, palpitations and precordial pain. Occasionally the tachycardia persists for months. While I have no observations on this point, it is quite likely that hypotension will be of frequent occurrence as a post-influenzal condition, especially in those cases with marked nervous exhaustion.

Venous thrombosis may occur. The veins most frequently involved are those of the lower extremities. Personally, I have seen no examples of this.

Nervous System.—The effect of this disease on the nervous system is marked. The most frequent of the nervous manifestations are *psychasthenia and neurasthenia,* which as a rule occur together. In a patient subject to these conditions the convalescence is satisfactory except for the failure to regain strength. He feels weak, tires easily and the slightest exertion produces a tachycardia and sweating. In addition there is a feeling of depression and an inability to perform the slightest mental effort. In extreme cases the despondency may be severe enough to cause the patient to attempt suicide. Recovery from these conditions is the rule.

Neuritis may occur as a post-influenzal manifestation. It may take the form of a polyneuritis or simply effect a single nerve.

Encephalitis (epidemic or lethargic encephalitis, nona).—Following the epidemic of 1889–90 a number of cases of influenzal encephalitis were reported. In many of these there was also a monoplegia or hemiplegia. Among the cases reported since the recent epidemic instances of paralyses, ther than those affecting the eye muscles, have been uncommon.

The condition is characterized by marked lethargy, asthenia, vertigo, eadache, photophobia, diplopia and some alteration in the mental state. ever is usually present and lasts from two to five days; this is apt to be llowed by a subnormal temperature. The onset is usually characterized y the symptoms given above. Later the patient lies motionless in bed ften unable to make any voluntary movement. The face is usually xpressionless and mask-like, resembling that seen in Parkinson's disease. acial paralysis may be present. The most frequent form of paralysis s an ophthalmoplegia which is noted in about 75 per cent. of the cases. he speech is often distinctly altered. Sentences are slowly uttered nd the words slurred; or the words may be uttered with great rapidity.

The spinal fluid may be under pressure but as a rule shows nothing bnormal. In a few cases the cell count has been increased.

Serious mental disorders may occur also as post-influenzal manifestations. Burr states that these mental disorders may be: (1) A state of mild elation without delusions (very rare); (2) confusional insanity; (3) dementia præcox, alcoholic hallucinosis and (rarely) paresis. In the last named group the influenza is not the cause but the excitant.

Gastro-intestinal System.—Digestive disturbances may persist and unduly prolong the convalescence because of malnutrition. There may be considerable loss of weight as a result. Long after recovery from the influenza patients may continue to suffer from recurrences of gastro-intestinal symptoms or gall-bladder trouble.

Diagnosis.—When epidemic influenza first makes its appearance it has been the common experience that the first cases are not recognized but are usually looked upon as severe "colds." Once the disease becomes prevalent, however, its recognition is not difficult. The condition I have experienced the most difficulty with is *typhoid fever*. Several sporadic cases of the latter disease were admitted to the University Hospital during the height of the epidemic and for some days their true nature was not recognized. This was due to the fact that headache, fever and bronchitis are common to both conditions. The mistake is less likely to occur in communities where typhoid fever is endemic and where one is constantly on the alert.

The real difficulty is not so much in recognizing the case as one of influenza as in determining the presence of pulmonary or pleural complications. Bearing in mind that influenza itself is usually a relatively brief infection the prolongation of the fever and respiratory symptoms beyond a week point strongly to some complication or extension of the inflammatory process to the pulmonary tissues. The recognition of these various conditions has already been dealt with.

PULMONARY FIBROSIS

This condition is variously designated as *chronic pneumonia, interstitial pneumonia* and *cirrhosis of the lung.* In the older literature on the subject the term fibroid phthisis is frequently used. The latter term is misleading, however, and should never be applied to this affection but should be restricted to those cases in which the tubercle bacillus is the exciting cause of the fibrosis.

Fibrosis of the lungs is of interest because of the similar effects and symptoms produced by a lesion which is the common result of many different processes. Properly speaking, it is not a disease; it is simply the result of some previous pulmonary affection. While the etiological and pathological features of pulmonary fibrosis are extremely varied the clinical picture is fairly characteristic and it is for this reason that cases of this nature are grouped together.

Etiology.—The etiology of fibrosis of the lungs embraces practically every disease to which the lung is subject, whether acute or chronic. The chronicity of the primary affection, however, undoubtedly has a great deal to do with the process. Thus, while it is recognized that the acute pulmonary affections, as broncho-pneumonia or croupous pneumonia, may terminate in an interstitial pneumonia, it is the chronic affections that furnish the greatest number of examples of the condition.

Of the chronic pulmonary infections tuberculosis is generally regarded as being the most potent factor in the production of fibrosis. In the majority of cases the tuberculous origin of the fibrosis can be determined by the presence of tubercle bacilli in the sputum. Occasionally, however, as in a case observed at the Phipps Institute, tubercle bacilli are not detected during life and the true nature of the fibrosis is revealed only by a histological examination of the lungs.

ne of the most frequent of post-mortem findings is a small indurated in one or the other of the pulmonary apices. Such areas are believed evidences of a healed tuberculous focus although there is no definite that such is always the case as most of them show neither tubercle i nor any histological evidence of tuberculosis.

he next most potent factor in the production of pulmonary fibrosis st. Under these circumstances the fibrosis is bilateral and diffuse. aspect of the subject has been dealt with fully in the chapter on moconiosis.

he acute inflammatory affections of the lungs furnish relatively few ples of the condition.

mong the rarer causes of pulmonary fibrosis may be mentioned ilis, abscess, infarct and the mycotic infections. Chronic passive stion sometimes leads to interstitial changes, known as "brown ation." This is an unusual cause. In such instances the lung nly shows an overgrowth of fibrous tissue but, in addition, it is brownish red color due to the deposit of hemoglobin.

n addition to the intrapulmonary affections, extrapulmonary condi- , as aneurisms, mediastinal growths, and pleural effusions may uce an overgrowth of fibrous tissue. The importance of pleural ions, especially when purulent, has been underestimated. An empy- is not an infrequent sequel of pneumonia and very often it is encysted. e effusion is not detected and removed it acts as a foreign body and ason of the pressure it exerts on the pulmonary tissue, leads to inter- l changes.

Iorbid Anatomy.—Fibrosis of the lung presents an extremely varied arance pathologically—a fact that has had not a little to do with liversity of names under which the condition has been described. one of the several parts comprising the lung may be the starting of the fibrosis: the tissue about the bronchi and blood-vessels, the lobular septa, the alveolar walls, or the pleura. When the process een of long standing, all of these structures may become involved, any trace of the original starting place be obliterated.

he varied conditions under which the fibrosis may arise and the ominance of the lesion in one or the other of the structures first ked render a satisfactory classification impossible. For the sake nvenience most writers recognize a *local* and a *massive type*. Local- areas of fibrosis are often seen. The most common example is that equently encountered at the extreme apex of the lung.

he massive type is of the most interest, both pathologically and cally, and the great majority of cases seen and recognized as fibrosis ng to this group. The massive type of fibrosis may be further di- d into a tuberculous and a non-tuberculous form. While the ulti- clinical picture is frequently identical in the two forms, the early es differ.

n the tuberculous form the process starts at the apex, while fibrosis ng from other cause most frequently has its origin at the base or of the lung. This is of considerable importance in differentiating two forms when tubercle bacilli are absent. It must be borne in l, however, that non-tuberculous fibrosis of an upper lobe does occur, when associated with bronchiectasis is often clinically indistinguish- from healed tuberculosis with cavity formation—a point Jacobi has

especially emphasized (see Fig. 221). In either case the dis
unilateral, the affected side is sunken, the spine bowed, and the sl
depressed. On opening the chest the heart is seen to be drawn
the affected side and the unaffected lung voluminous.

The organ on removal is found to be hard and airless and s
resists cutting. In the extreme grades the entire lung may be red
a mass of fibro-cartilaginous tissue with dilated bronchi. Betwe
and the lesser degrees of involvement all gradations exist. The

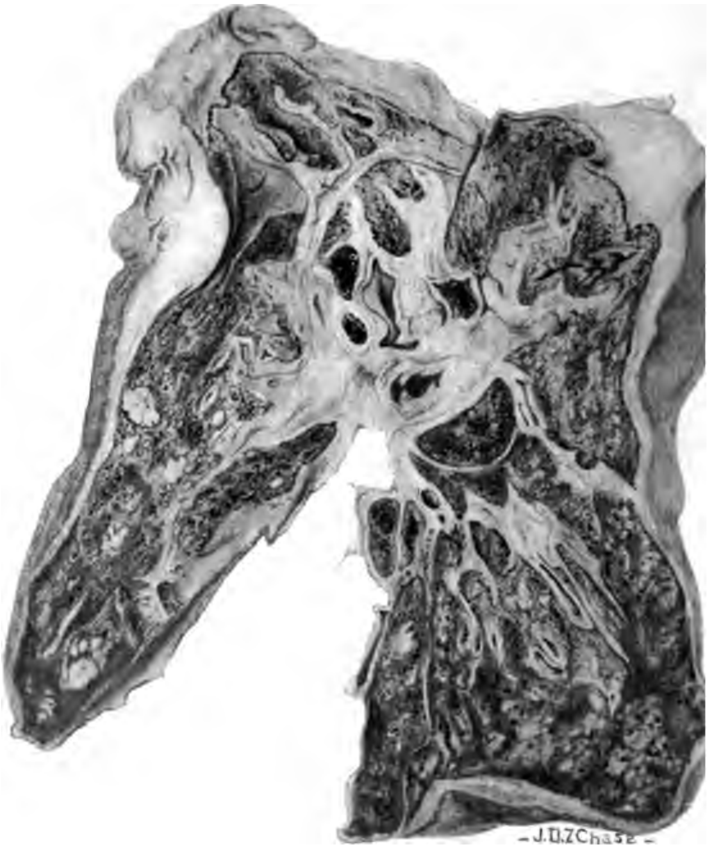


Fig. 299.—Fibrosis of the lung in which the greatly thickened pleura and the fibr
root of the lung are independent of each other. (*Phipps Institute, Fifth Re*

involvement is distinguished by the areas of grayish tissue, whi
be most prominent at the root of the lung, with radiating bands
ing toward the periphery (Fig. 299), along the interlobular septa
the bronchi and blood-vessels. Fig. 300 represents a type of
described by Kidd and W. McCollum, and termed by them r
In this specimen the lung is seen to be intersected by fibrous
which follow the interlobular septa. This specimen also sh
embedding of the bronchi and blood-vessels in the masses of
tissue.

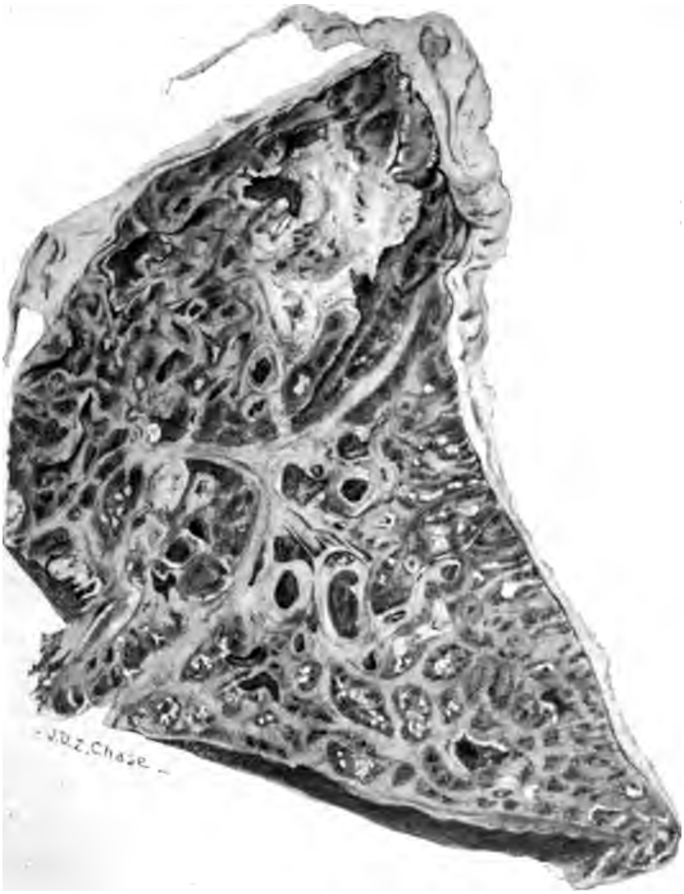


FIG. 300.—Reticular fibrosis. (*Phipps Institute, Fifth Report.*)

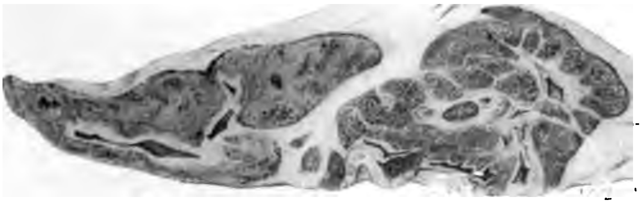


FIG. 301.—Fibrosis of the lung in which the thickened pleura and the fibrosis within the lung are definitely associated. (*Phipps Institute, Fifth Report.*)

An almost constant feature of these unilateral cases is the associate pleural thickening. The pleural cavity becomes obliterated and th lung is bound down by dense adhesions, so that it is removed from th chest cavity only with the greatest difficulty. As a rule, fibroid change in the pleura form a part of the general process and depend on the sam

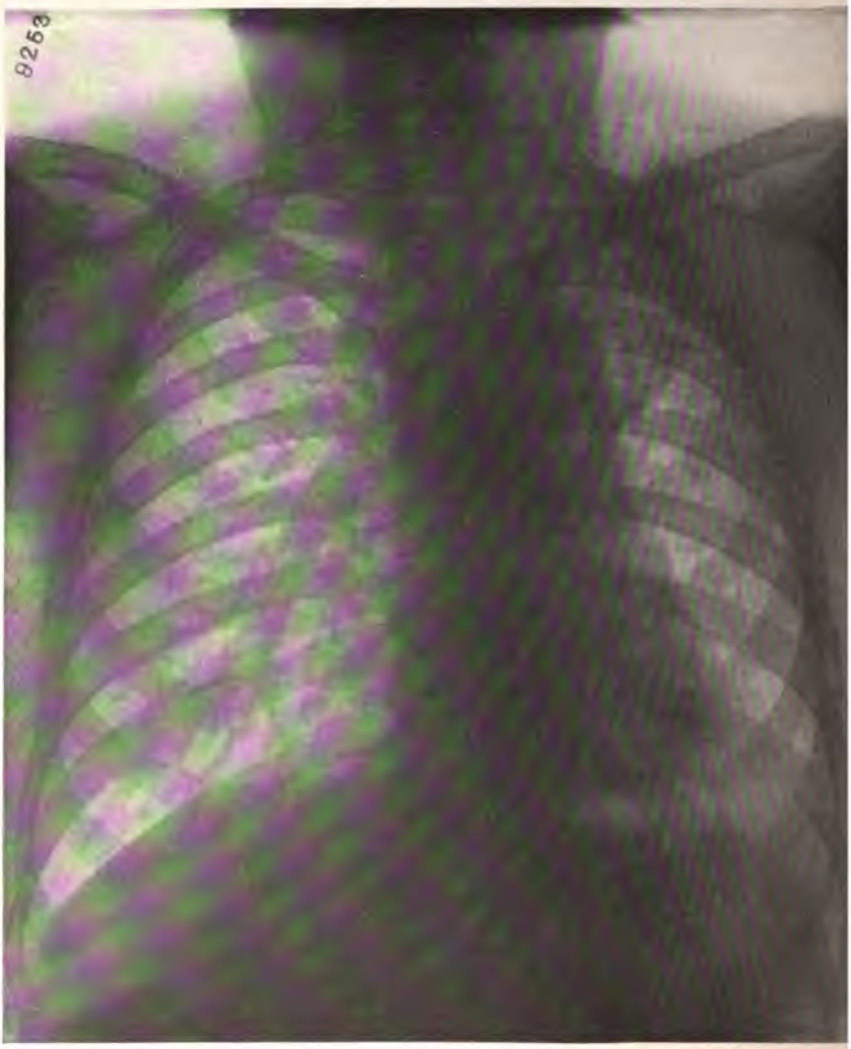


FIG. 302.—Chronic fibrosis of left lung. Marked retraction of left chest. Heart a great vessels displaced toward the affected side.

exciting cause as the intrapulmonary change. Whether the fibro hyperplasia can begin in the pleura and by extension inward along t interlobular septa secondarily involve the lung is not clear. As can seen in Fig. 301, the greatly thickened pleura and the fibrosis at the ro of the lung are independent of each other, while in Fig. 302 the thickened pleura and the fibrosis within the lung are definitely associated. It is quite probable that in some instances the two processes are dependent on the same exciting factor. Rarely the pleura may escape entirely.

It is not often that a lung which has undergone fibroid change retains its normal size. Such a lung is nearly always smaller than normal, the reduction in size being, in some instances, extraordinary. In the variety known as Corrigan's cirrhosis the lung may not exceed the size of a clenched fist.

Dilatation of the bronchi is pretty constantly present in extreme grades of fibrosis. In some instances the bronchial dilatation is the most prominent feature (see section of Bronchiectasis).

Fibrosis of the lung secondary to croupous pneumonia is not common. Occasionally the fibrinous exudate is not removed and a fibrous hyperplasia may start in the septa and peribronchial tissue. In fibrosis of this type the lung is red in color, tough and airless, and presents the appearance of a piece of muscle. This constitutes what is known as carnification of the lung.

When the fibroid process is tuberculous in origin, apical cavities are commonly present and the fibroid tissue is densest about the cavity. The overlying pleura also becomes greatly thickened. Gradually the process extends until the rest of the lobe or entire lung is involved in the fibroid change. In one case at the Phipps Institute the enveloping layer of fibrous tissue about an apical cavity was at no point less than 3/4 inch in thickness. The fibrous tissue at times becomes cartilaginous and very hard.

The heart is usually displaced toward the affected side (Fig. 302) and shows hypertrophy or dilatation of the right ventricle. The findings in the other organs do not conform to any special rule. Frequently there is evidence of arterio-sclerosis, interstitial changes in the kidneys, and occasionally in the other viscera. This widespread sclerosis in the various viscera led at one time to the belief that the pulmonary fibrosis was simply a part of a general fibroid diathesis. Such a view no longer receives any support. The age of the patients and the congestion which results from the obstruction offered to the right heart are doubtless the true causes of the associated fibrosis.

Symptoms.—The clinical picture of massive fibrosis due to the tubercle bacillus and that which results from a chronic inflammatory condition is identical. Whether the fibrosis is due to tuberculosis or not in a given case must depend to a great extent on the examination of the sputum. In the majority of instances the patient is past the middle period of life when first seen. While fibrosis due to the tubercle bacillus is often under observation during the period in which the pathological changes are taking place, one rarely sees the pure form of fibrosis until the process has become well established. Patients presenting either form of fibrosis give a history of long-standing cough, expectoration, occasional attacks of blood spitting and dyspnea on exertion. The temperature is either normal or shows only a slight afternoon rise (99°F.). The pulse rate is not accelerated. In spite of the long duration of the disease, from five to twenty years or more, the general health may be but little impaired. The patient suffers no great inconvenience, except dyspnea on exertion and takes part in the ordinary affairs of life.

So long as the cardiac functions are normally maintained the patient may not consult a physician. When first seen it is usually because of an attack of pleurisy, of blood spitting or because of increased cough, dyspnea or swelling of the feet, the latter symptom being indicative of failing compensation. The exceptions to this general rule are the cases in which dilatation of the bronchi is a prominent feature. If bronchiectasis is present, the sputum is usually very abundant and not infrequently has a very offensive odor.

Physical Signs.—*Inspection.*—The affected side is shrunken and motionless and the shoulder on this side lower than its fellow (Fig. 302). The intercostal spaces are obliterated, and in some instances the ribs overlap. The spine is bowed laterally, the convexity being always *away* from the affected side; the angle of the scapula may be tipped back from the chest wall. Litten's shadow is absent on the affected and well marked on the unaffected side. A fluoroscopic examination will show the diaphragm to be much flatter than normal on the affected side. The chest tracing in Fig. 303 shows the great disparity existing between the two sides of the chest.

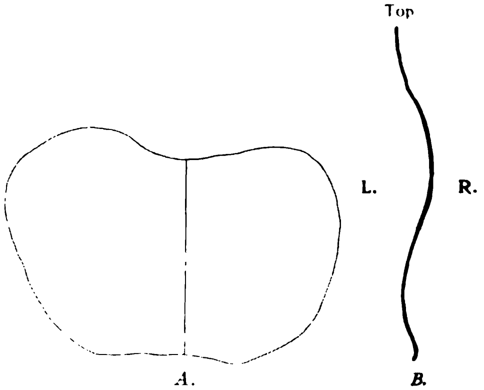


FIG. 303.—(*A*) Chest tracing from a case of left-sided fibroid disease. (*B*) Spinal deformity showing marked curvature of spine away from the affected side.

Displacement of the heart is an almost constant feature. When the left side is affected, the heart is pulled upward and outward and may be displaced to the fourth interspace in the mid-axillary line. In right-sided cases the displacement is even more striking. Strong pulsation may be noted in the fifth or sixth interspace in the right nipple line. It is to be borne in mind that the pulsation in this situation is not caused by the apex but by the body of the heart.

The unaffected side is full, the interspaces wide, and the expansion free.

Marked clubbing of the fingers and also of the toes may be present if the bronchi are dilated.

Palpation.—This serves to verify the diminution of expansion and the location of the apex beat. Over the affected side the tactile fremitus varies; it may be greatly diminished or it may be exaggerated.

Percussion.—Over the affected side, especially over the base of the lung the percussion note is impaired or dull. If dilatation of the bronchi a prominent feature a tympanitic percussion note may be elicited near e angle of the scapula or if the upper lobe is involved, beneath the ıvicle. Over the unaffected side the note is hyperresonant. Topoaphical percussion of the healthy lung will also show that it extends yond the middle line anteriorly and that a resonant note is obtained inch or more below the normal base line posteriorly.

Auscultation.—The breath sounds over that portion of the lung which s undergone fibroid change are often distant and the expiratory sound ay have a prolonged and bronchial quality. If the bronchi are dilated, nphoric breathing and whispering pectoriloquy may be heard near the ıgle of the scapula. Râles may be absent. If present they may be of ıe fine cracking variety or they may be wheezing in character. Vocal sonance may be diminished or exaggerated.

Diagnosis.—The salient features of massive unilateral pulmonary brosis are: a history of long-standing cough, dyspnea and marked etraction of one side of the chest with displacement of the heart toward he affected side. If the lower lobe is involved and the upper one is elatively clear, the condition, in all probability, is not due to tuberculosis. Vhen, however, the upper lobe is the site of the change and, in addition he bronchi are dilated, pure fibrosis cannot be differentiated from that lue to tuberculosis, without a sputum examination. The neglect of this recaution leads to many mistakes.

In regard to pulmonary fibrosis and *bronchiectasis* it is not a question f distinguishing between them, but of determining whether the two conlitions are associated. Both are due to the same causes. In about 80 per cent. of the cases of massive fibrosis the bronchi are dilated. Rarely the bronchi are dilated without associated interstitial changes in the pulmonary tissue. If in a case with marked unilateral retraction of the chest wall there are also present a paroxysmal cough, profuse expectoration, often of a foul odor, and marked clubbing of the fingers, it is reasonably certain that in addition to the fibroid changes, the bronchi are also dilated.

One of the most frequent errors is that of mistaking a chronic *empyema* for a chronic pulmonary affection. In the early stages of a purulent effusion the effected side is usually fuller than the unaffected side. If the empyema goes unrecognized, it sometimes undergoes partial absorption. The liquid portion is absorbed leaving behind a putty-like mass made up of pus cells and lymph. In addition the pleura becomes greatly thickened. As a result the chest wall becomes retracted and eventually the lung itself undergoes interstitial changes. I have already alluded to the part played by encysted empyema in the production of pulmonary fibrosis.

CHRONIC INFLAMMATORY CONDITIONS OF THE LUNGS OF UNCERTAIN ETIOLOGY

In the course of a year there are seen in every hospital and dispensary evoted to the treatment of chest conditions, a number of cases in which he lesion is most marked or limited to one or both bases of the lungs. ı the majority of cases the lesion is unilateral.

A number of these cases have been reported in the literature as exnples of ***subacute*** or ***chronic pneumonia.*** They lack the characteristics of definite pulmonary fibrosis in that there are often no marked structural changes in the lungs; the chest wall is not retracted and the physical signs are rarely as marked as in pulmonary fibrosis. There are good reasons for believing, however, that the condition under discussion is often the preliminary stage of fibrosis and bronchiectasis as both of the later conditions so commonly give the same indefinite history as to onset.

Of seventeen cases of this nature seen at the Phipps Institute during the past three years nearly all have been mistaken for tuberculosis by reason of the fact that they complained of cough, expectoration and occasionally had had blood-streaked sputum or even small hemoptyses. This also seems to have been the experience of sanatoria physicians who have reported cases of this nature.

My experience has been that a patient with this condition almost invariably presents himself for treatment because of a persistent cough, usually a considerable amount of muco-purulent expectoration, and occasionally blood-streaked sputum, or a small hemoptysis. Some of them also complain of shortness of breath. There is no fever, no acceleration of the pulse-rate and little or no loss in weight. In most instances the cough has been present for a year or more. The symptoms, of course, may readily be those of pulmonary tuberculosis but on examining the chest it is found that the apices are free from any abnormal signs. At one base, occasionally at both, the breath sounds may be slightly suppressed. The principal finding, however, is the presence of râles, which may be of the fine subcrepitant type or they may be mixed. In not a few instances the râles are so numerous that the breath sounds are almost entirely obscured.

That the condition is often secondary to an antecedent inflammation of the pleura, with or without an effusion, is suggested by the fact that the diaphragm is so frequently fixed and that the roentgenogram shows some haziness over the affected area. Another possible exciting cause is the presence of a chronic abscess of the lung. When this condition exists it acts as a foreign body and thus tends to lead to the promotion of fibrous tissue.

In the majority of the cases we have seen at the Phipps Institute, the condition has been so vague in its onset that it has not been possible to determine the exciting cause. As a sequel to the recent epidemic of influenza I have seen several cases in which there was a persistence of râles limited to one base. The explanation in these cases would seem to rest on the assumption that it is well known that influenza broncho-pneumonia frequently involves the interstitial tissues and that such lesions clear up very slowly. Whether the râles in the cases I have referred to will eventually disappear or persist is problematical at the present time. A marked increase in the number of cases of this nature is to be expected in view of the fact that interstitial changes in the lungs are common in the broncho-pneumonias caused by both the influenza bacillus and the *streptococcus hemolyticus.*

The bacteriological findings are varied and no one organism occurs alone or even predominates. It is needless to say that tubercle bacilli are absent.

The X-ray examination of these cases is, in the great majority of instances, very unsatisfactory. In spite of the fact that the physical signs are marked it more often happens than not that the roentgenogram shows nothing or only slight and indefinite shadows. This experience is confirmed by others who have reported similar cases. When dyspnea is at all marked the diaphragm is usually found to be partially or completely fixed on the affected side.

Of the seventeen cases referred to the left lower lobe was involved eight times, the right lower lobe six times, and both lower lobes twice. The diaphragm was fixed on the affected side in five instances.

Although, as I have pointed out, the condition is commonly mistaken for tuberculosis, there is no excuse for this. As I have repeatedly emphasized tuberculosis is never primarily a basal condition. The fact that the physical findings are entirely limited to the base of the lung therefore excludes tuberculosis at once. Limitation of the physical signs to one base, especially the right, in children should always suggest the possibility of a foreign body.

CALCIFICATION OF THE LUNGS

The occurrence of calcareous deposits in the lungs is not uncommon. As a rule, they are found near the apex and represent what was once a small cluster of tubercles which had been surrounded by fibrous tissue and later by the deposition of lime salts (Fig. 229). Occasionally these nodules become disintegrated and are expectorated in small particles at a time. They are usually grayish white in color, gritty and hard but may be of a putty-like consistency. They vary in size, ranging from that of a pin-head to that of a hazel nut. They may be smooth and round or very irregular and rough. They are commonly referred to as lung stones or pneumoliths and, in the great majority of cases, are encountered in phthisical individuals.

Helbig[1] reports the case of a woman who spat up a number of these lung stones which on microscopic examination were found to contain bone corpuscles and lamellæ. In this connection may be mentioned a case observed by Mangini[2] in which bone needles could be felt in the lower lobes and cohering to form an actual arborization. He found records in the literature of 23 cases of this ossifying nature. The process is believed to result from repeated inflammatory processes. It is quite possible that there may have been an initial deposit of calcareous material which was finally converted into osseous tissue.

These local calcareous deposits, so frequently seen in the lungs of tuberculous subjects, may also occur in other situations, namely—in the arteries of old people and in pleural or pericardial exudates where extensive calcareous plates may form. There may also be a deposit of these salts in tendon sheaths, bursæ, etc. In addition to these localized deposits extensive and multiple calcareous metastases may occur. Wells[3] has reported some 36 cases of this nature. The lungs, endocardium of the left ventricle and the pulmonary veins seem to be especially predisposed t this change in association with bone tumors, osteomalacia and l remic conditions.

arbitz[4] in reporting a case of extensive calcification of the lungs states that the condition may be metastatic, dependent on bone destruction, or, it may arise without any indication of bone disease. In the case reported by Harbitz the lungs weighed nearly six times as much as normal. The lungs could be cut only with difficulty and felt like porous bone. Passing the finger over the surface felt like rubbing it over sand-paper, and here and there were round or irregular concretions. The distribution of the mineral matter was about the same in both lungs. The cut surface was of a peculiar, reddish-brown color with numerous small holes in the extensive stroma. After drying a small slice of lung substance it had a peculiar yellow-red color and a porous surface which felt like sand-paper and from which calcareous granules fell out. The deposit of calcareous material is, however, not always so extensive as in the case observed by Harbitz. The process may be confined to one lung or only a portion of one lung.

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[1] *Münch. Med. Wochens.*, Oct. 17, 1916.

Lyon Medical, April 26, 1914.

rchives of Internal Medicine, 1911, 7, 721; *Ibid.*, 1915, 15, 514.

hives of Internal Medicine, Jan., 1918.

There is nothing distinctive in either the symptoms or the physical signs. Dyspnea, cyanosis, cough, and eventually edema of the extremities, due to failure of the right heart, have been the principle symptoms noted. The condition might be suspected in the case of an individual suffering from some destructive bone lesion or in case the roentgenogram showed the presence of unusually dense shadows.

PNEUMOCONIOSIS

The term pneumoconiosis (πνεύμων, lung; ἡ κονις, the dust) is applied generally to pulmonary affections which develop as the result of the inhalation of dust. "The term 'dusts' as I would here apply it, includes all those fine, solid particles which are thrown off from various substances in the processes of manufacture or treatment of articles in common use in daily life, such as earthenware utensils, knives, needles, or mechanical instruments, like files or saws; or ornamental things, such as ornaments of pearl, ivory or turned wood; or articles that are worn, of silk, cotton, hemp, fur; or things that we use for food, such as flour; or for creating warmth such as coal; or for using as a supposed luxury, such as tobacco and snuff."[1] From the above description it is seen that the term pneumoconiosis is a very comprehensive one. A number of synonyms have been used to indicate the particular variety of dust, namely, anthracosis or coal dust; chalicosis or flint dust; siderosis or iron dust; byssinosis or cotton dust; tobacosis, etc. In addition a variety of popular names have been applied to the dust diseases, the terms used indicating the occupation, as for example, miner's asthma, grinder's rot, potter's rot, potter's asthma. That the prolonged inhalation of dust is capable of producing pathological changes in the lungs and bronchi has been long known. For the first systematic treatise on the subject we are indebted to Ramazzini (1703). During the last century the subject was thoroughly studied, especially by English physicians, who had unusual opportunities because of the great diversity of manufacturing processes in England and the large number of people engaged in these trades. In this country the subject of occupational diseases has received but scanty attention and it is only within the past few years that several of the States have required physicians to report on their occurrence.

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[1] RICHARDSON, *Scientific American Supplements*, 1876.

Etiology.—The etiology of pneumoconiosis is very complex. The dition is caused by a variety of dusts, some of which are exceedingly ating and produce in a relatively short time, marked damage to the iratory tract; in the case of others, pulmonary changes are produced slowly and the general health remains unimpaired for years. Furmore, individuals seem to vary in their ability to withstand the cts of the same kinds of dust. Some workers after years of prolonged osure show little or no impairment of health while others, at varying ods, gradually succumb to its effects. In addition there are other perating factors, not connected with the occupation itself, such as an nitary living environment, unhygienic home, insufficient food, and mperance. This latter aspect of the subject has been emphasized a number of English writers on the subject. In the case of miners great depth at which some of them work may have some influence in nging about respiratory changes.

The extent to which pneumoconiosis prevails cannot be determined the term is practically never employed to indicate the cause of death even to indicate the nature of the illness during life. How many es of bronchitis, acute or chronic, of broncho-pneumonia or other acute d chronic inflammations of the respiratory tract are produced as the ult of the inhalation of dust, is therefore problematical. The only ception to this is tuberculosis. For many years it has been recognized at the mortality from tuberculosis among those working in the dusty des is excessive.

There are two main types of dust: (1) the inorganic, and (2) the ganic.

Inorganic Dust.—This is by far the most harmful because of the arp, angular particles which produce harmful mechanical injuries and cause, in some instances, injurious chemical effects are also produced. he different forms of pneumoconiosis are as follows: (*a*) Anthracosis niner's asthma, coal miner's phthisis, black spit). This is by far the mmonest form of dust disease and the one about which the most has en written. The condition exists to some extent in practically all who ve worked for any length of time in a coal mine. It occurs in a more vere form in those who mine the anthracite or hard coal. This is due the fact that the bituminous or soft coal is not hard and stony as the nthracite.

The lungs of practically all urban dwellers show some evidence of nthracosis. Usually it consists of nothing more than the grayish disloration of the lungs common to all who reach adult life but in communities in which the amount of coal smoke and soot is excessive the ndition becomes more marked and may constitute a true anthracosis. otz[1] in a recent study has shown that among those who have lived for ears in Pittsburgh, Pa., the amount of carbon deposited in the lungs is cessive as shown both by the residual ash and the degree of pulmonary brosis produced.

Chalicosis and Silicosis.—In these forms of pneumoconiosis the dust onsists of sharp angular particles which give rise to considerable echanical injury to the mucous membrane. Aside from dust which ontains poisonous mineral substances, such as lead, that which is composed of fine particles of flint or silex is the most dangerous form encountered. Flint and silicious dust are met with in a variety of occupations, namely, potters, stone masons, stone quarry workmen, etc. Among the gold miners of South Africa a particularly severe form of infection occurs from exposure to dust composed of fine particles of silex. The dust arises as the result of using machine rock drills (Oliver).

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[1] *Am. Jour. Public Health,* Oct., 1914

Siderosis.—This is due to fine particles of iron dust. The condition is rare. The occupations involved are those in which the red oxide of iron is used. Looking-glass makers, gold beaters, and glass polishers are among the workers exposed to this form of dust. In cases of this type the sputum may be reddish and at post-mortem the lung may present a brick-red appearance due to the red oxide of iron. Among workers who smooth or sharpen iron or steel implements on a sandstone, pneumoconiosis is quite common. This form is due, in part, to fine particles of metal and, in part, to fine particles from the sandstone.

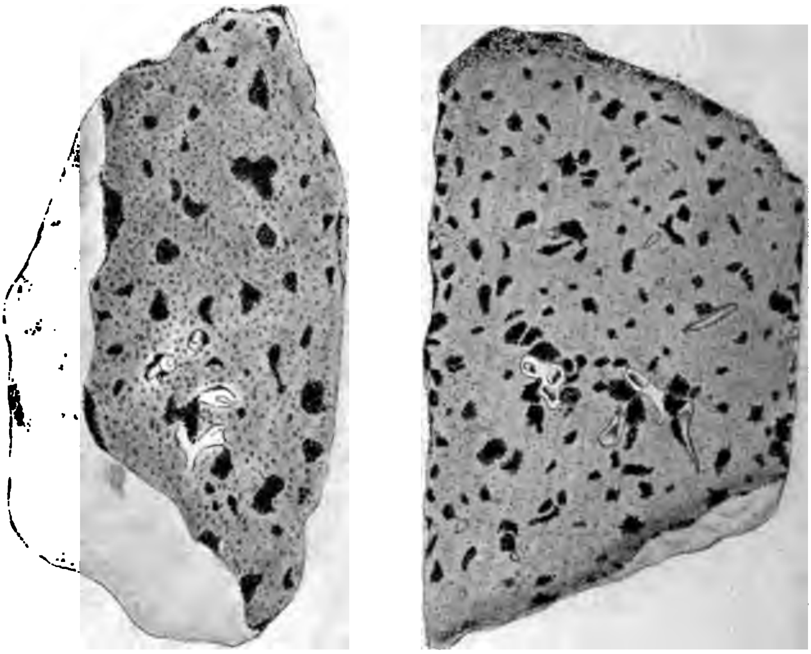

FIG. 304.—Nodular dissemination of anthracosis. (*Klotz, in American Journal of Public Health.*)

Dust from Crushed Slag.—Slag, a name applied to the refuse from blast furnaces after the iron has been removed, is sometimes finely crushed. In this pulverized form it is used as a fertilizer because of the large quantity of phosphate of lime which it contains. This form of dust produces a curious infection of the lungs in that it resembles acute croupous pneumonia. This form of infection will be considered more in detail under the section on Morbid Anatomy.

Organic Dust.—The evidence that inorganic dust is capable of roducing definite pathological changes in the lungs is conclusive. When e come to consider the effect of organic dust, however, we lack definite roof that it is of itself injurious. While it is true that many of the ccupations in which organic dust occurs are attended by a high death ate from tuberculosis, it is questionable whether the dust plays anyhing more than a very minor rôle. Oliver states that if we leave out f consideration the moisture which has been artificially introduced in he weaving sheds, it cannot be said that the manufacture of cotton is *er se* an unhealthy occupation. In this country much of the tuberculosis and general ill health among cotton operatives can be traced to heir deplorable living conditions, and to the fact that many especially n the South, enter the trade at an early age.

In the manufacture of felt hats the hair of rabbits and similar animals s largely used. This occupation has been classed among the dangerous dusty trades. At the Phipps Institute it has been our experience that among the hatters who applied for treatment all were employed in the molding room which is wet and damp. Oliver states that he has never seen ill health among those engaged in the handling of tobacco, which could be attributed to the trade itself. In the United States cigar makers have a very high mortality from tuberculosis. This is due, in all probability, to the fact that the workers are rendered more susceptible to this infection because of a deterioration of health due to nicotine poisoning. Workers in this trade have informed me that many of them are rejected for insurance because of a "tobacco heart."

It is my personal belief that pneumoconiosis rarely if ever develops as the result of exposure to organic dust. Not only is this form of dust incapable of producing a mechanical injury, but it is doubtful if any of it ever reaches the finer bronchi and lung cells. The vast majority of the dust particles are prevented from penetrating very deeply into the bronchial tube because of the action of the ciliated epithelium and the moisture in the larger tubes. The high incidence of tuberculosis among those exposed to this type of dust is to be explained in other ways, namely, low wages, bad home environment, poor food, etc.

During the *threshing season* in the fall, it is not uncommon for those who are exposed to the dust produced by the wheat, oats, etc., passing through the threshing machine, to develop acute symptoms. Workers who are especially susceptible will, in the beginning of the threshing season, develop a headache and great irritation of the respiratory tract. In addition there is considerable depression, a sensation of chilliness and often high fever. After a few days these symptoms pass off and the worker is apparently immunized for the remainder of the season. These symptoms have been attributed to a pollen infection, especially that of rag weed, and are believed to be somewhat analogous to the autumnal form of hay fever. A similar condition is encountered among those engaged in the manufacture of shoddy. During the grinding of the rags by machinery considerable quantities of dust are thrown off, inhalation of which is extremely trying to new hands but which has little or no immediate effects on the older work people. Those persons who are new to the trade develop what is known as "shoddy fever," the sympoms of which are a rise of temperature, severe headache, signs of bronhial catarrh, and running at the nose. The work people shiver as if they were going to have a severe fever, and they complain of muscular pains (Oliver).

Pneumoconiosis and Its Relation to Tuberculosis.—The relationship which exists between the inhalation of dust and tuberculosis is an interesting one, and the belief that dust has a strong predisposing effect in producing pulmonary tuberculosis is quite generally accepted. When we recall, however, the changes which are produced in the lung by reason of the inhalation of inorganic dust, it is evident that the process is in the beginning, a non-tuberculous fibrosis of the lungs. Furthermore, it is well known that the symptoms and physical signs produced by such changes often simulate those produced by tuberculosis very closely. It seems quite likely, therefore, that in not a few instances it has been assumed that the process was tuberculous, and death was ascribed to this cause, without an examination of the sputum having been made to determine the true nature of the disorder. Tatham[1] in commenting on this aspect of the question states that: "Potters succumb to non-tubercular disease of the lungs much more rapidly than they do to tubercular phthisis, and it is certain that much of the so-called potter's phthisis ought properly to be termed cirrhosis of the lung. Deaths from this affection should never be included under the head of phthisis, which term is now restricted by universal consent to the tubercular malady of that name."

On the other hand, it is to be borne in mind that the cause of death in cases of this type is not infrequently given as being due to chronic bronchitis, emphysema, pleurisy, etc. This might possibly equalize the error.

There is still another factor to be considered, namely, the effect of an antecedent fibrosis of the lungs. It is well known that the stimulation of connective-tissue growth is the way in which nature overcomes tuberculosis. Therefore, anything which stimulates such a growth in the lungs should aid in preventing the tubercle bacilli from getting a foothold, or in overcoming or retarding their growth, if they become established in the pulmonary tissue. This is the view held by Wainwright and Nichols, who found that true tuberculosis was not a frequent finding among coal miners, but that extensive fibroid changes in the lungs, as the result of coal dust, was exceedingly common. The protecting influence, in their opinion, was the fibroid tissue and not the coal dust itself, the latter having been held by some to have germicidal qualities.

While potters, for instance, are not freed from the danger of becoming tuberculous by reason of the fibrosis which is produced incidental to the inhalation of clay and flint dust, it seems reasonably certain that the fibrosis does tend to retard the progress of the tuberculous infection. Thus it has long been recognized that the age period at which potters succumb to tuberculosis, if it does develop, is far beyond the average, and in addition, so far as my observation goes, the disease seems to be much less severe than the same amount of damage produced in other classes of patients.

Morbid Anatomy.—While all forms of inorganic dust bring about essentially the same changes in the respiratory tract, they vary, one from another, in the extent of the lesions produced and the severity of the symptoms. The sharper and more angular the dust particles, the greater will be the amount of mechanical injury and hence the greater the inflammatory reaction.

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[1] OLIVER'S "Dangerous Trades."

The least dangerous of the inorganic dusts is that arising from coal, especially the soft or bituminous variety. It is this form of pneumoconiosis that has been most thoroughly studied. At the present time, it is pretty generally agreed that pneumoconiosis is the result of dust taken into the lungs during the inspiratory act. The view that the dust is conveyed to the lungs by way of the lymphatics after having been swallowed, is of little importance, as far as this type of infection is concerned.

Exposure to almost any kind of dust, even for a short time, is apt to irritate the upper respiratory tract and produce coughing. If the exposure is sufficiently long there develops a pharyngitis and later a bronchitis. Between simple irritation as the result of a brief exposure, and extensive tissue changes, as the result of prolonged exposure, varying degrees of pathological alteration may occur.

Clay and flint both contain very hard, sharp, angular particles of silex, which when drawn into the respiratory tract during inhalation are not dissolved by the bronchial secretions, but are deposited in the alveoli and smaller bronchi. For a varying length of time the inhaled dust is arrested in its advance by the mucous secretion in the bronchi, by the ciliated epithelium lining the tubes and by the action of the phagocytes. Sooner or later, however, these defensive forces weaken, and finally the dust passes into the lymph channels, and also along the finer bronchi, until it reaches the parenchyma of the lungs. As a foreign substance it then sets up a chronic inflammatory process. The initial deposits of dust occur for the most part in the posterior and inferior portions of the lungs.

Microscopial Changes.—The gradual evolution of anthracosis has been described by Wainwright and Nichols,[1] Haythorn[2] and Klotz[3] to whom I am indebted for most of the following description.

1. In the case of those who have worked in a coal mine but a few months some of the epithelial cells lining the alveoli become swollen and contain particles of coal dust. Sometimes a few larger desquamated cells containing much pigment are found in the alveoli lying loose with some detritus and free dust particles. Even in the early stage, dust particles are seen in the walls of the air vesicles and around the mall bronchi. In this stage there is no evidence of connective-tissue roliferation.

2. In those who have worked in the mines for several years the swollen ells containing dust particles are much more numerous and in addition here are present large mononuclear phagocytes filled with carbon particles. hese pigment-bearing phagocytes are also found in the interalveolar mph spaces, in the lymphatics of the interlobular septa, in those about e vessels, and beneath the pleura and in the lower layer of the bronchial ucosa.

As the accumulations of pigment gradually increase, they not only rm lines along the septa of the lobules and the vascular channels, but dular collections appear at the points of junction of the various lymph annels where small receptaculi are formed. These nodules become so ominent that they are readily felt by the finger, and at times the course of the lymph channel can be detected by the sensation of touch (Fig. 304).

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[1] *Am. Jour. Med. Sc.*, 1905, vol. cxxx.
[2] *Jour. Med. Res.*, December, 1913.
[3] *Am. Jour. Public Health*, vol. iv, No. 10.

At the points where the dust collects the fibrous tissue is arranged in concentric layers, so as to form small nodules. Between the layers of fibrous tissue and especially toward the periphery, the particles of carbon are especially numerous. The fibrous masses are well seen in the septa that run in from the pleura. In places considerable-sized black triangles with their bases on the pleura are seen, showing where the septum has been completely filled up with dust. The striking point in the whole picture is the extensive plugging and obliteration of the small and medium-sized lymphatics and the compression of the large ones. In the pleura and the peribronchial lymph nodes the picture is a similar one (Haythorn). In this stage the bronchial and mediastinal lymph nodes become enlarged. As the result of exposure to nearly any kind of dust the bronchial lymph nodes become black, in the case of coal miners, intensely so.

3. The third change noted by Wainwright and Nichols is important from the standpoint of prevention. They found that in the case of individuals who had formerly been miners, but who for many years had not followed that occupation, the lungs no longer showed signs of irritation. The swollen epithelium had subsided and again become normal, and neither the cells nor the alveoli contained dust. The deposits of dust in the alveolar walls, the septa and peribronchial tissue, however, still remained, as did also the connective-tissue thickening.

Macroscopic Changes.—There is usually some evidence of emphysema. This may be extensive or it may be limited to patches along the margins or scattered through the body of the lung.

In cases where the exposure to dust has been a matter of years, the lung is hard and more or less solid as the result of fibrosis. The dominant feature of pneumoconiosis is the fibrosis. The fibroid changes are diffuse and bilateral (Fig. 307) but at times a portion of one lung or the entire organ may become densely fibroid. The organ often cuts with some difficulty owing to the presence of gritty, fibrous nodules. The cut surface may be smooth but usually has a gritty feel on section. In the case of anthracosis the cut surface exudes a black, ink-like fluid (Fig. 305). In this form of fibrosis of the lungs the walls of the bronchi are often thickened and cylindrical dilatation of the bronchial tubes is also commonly present.

The pleural surfaces are usually adherent, especially at the bases of the lungs.

The association of tuberculosis and pneumoconiosis has already been considered.

The changes as described above are, for the most part, common to all forms of pneumoconiosis due to inorganic dust. The changes produced in the lungs of the gold miners of South Africa and in those exposed to slag dust are worthy of special mention. According to Oliver, *gold miners' phthisis* is especially rapid in its development. Gold miners on the Rand can follow the employment but for a few years; as a rule from five to ten years. Besides being produced in a relatively short time, the fibrosis is very extensive and gives rise to the most extreme dyspnea. The disease is brought about by the inhalation of a very fine form of dust composed of sharp-pointed particles of silex. This dust is produced by

g the rock with machine drills. The tendency to respiratory
lers is also increased by reason of the fact that the men are rapidly
orted from a warm, deep mine to the surface where the air is cold.
te of the evidence of extensive pulmonary changes Oliver was struck
e fact that these men presented a healthy, bronzed appearance.

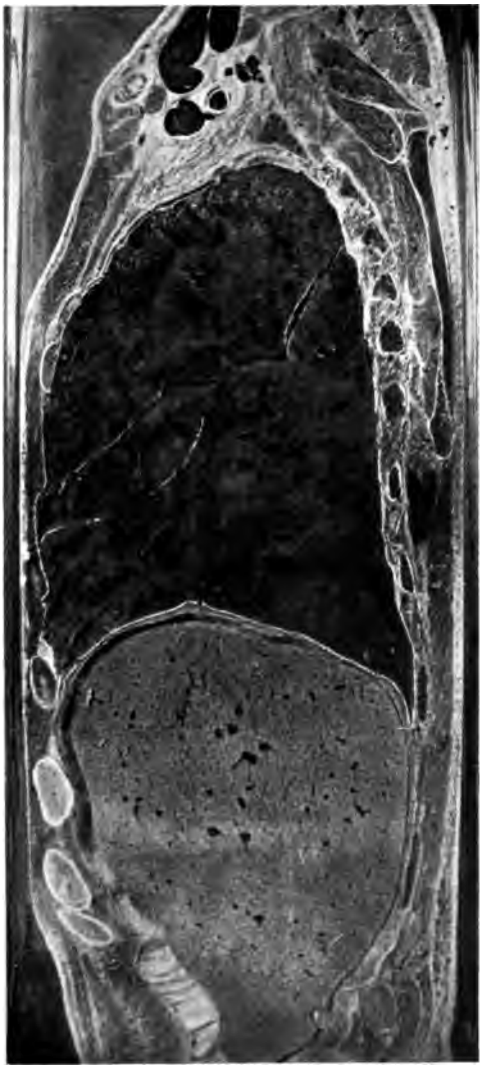

FIG. 305.—Anthracosis.

though those exposed to *slag dust* frequently develop pulmonary
e, the changes in the lungs are very different from that ordinarily
ntered in pneumoconiosis. The slag dust gives rise to an acute
mation of the lower lobe of one of the lungs, which after death is

found to be the seat of ordinary gray hepatization. "It is noteworthy that the inflammation of the lungs commences like pneumonia and runs a similar course, with rigor, pain in the chest, rusty sputa, and, in 30 per cent. of the cases, herpes on the lips. On physical examination signs of pulmonary consolidation are to be noted" (Oliver).

Symptoms.—Exposure to any form of dust, even for a brief period of time, is apt to cause considerable irritation of the upper air passages, as manifested by cough and sometimes sneezing. Owing to the fact that coal dust is relatively the least dangerous of the inorganic dusts, years may elapse before symptoms arise which are indicative of serious pulmonary damage. Among potters, serious symptoms rarely arise until the worker has been in the trade from ten to twenty years. According to Oliver the "gold miner's phthisis" of South Africa is the most serious of the dust infections as the average time the occupation can be followed is six and a half years.

In the dusty trades little or no inconvenience is experienced, in the beginning, from the inhalation of dust. As a rule, the first thing to be noticed is a slight irritation in, and a desire to clear, the throat. Next, a slight cough develops which may occur in the morning only. Later some blackish, viscid mucus appears after the cough. As the process advances, the cough and expectoration become more and more marked.

At some period, varying with the character of the dust, the worker begins to experience some tightness in the chest, the breathing gets shorter and shorter and finally the disturbed lung function becomes so pronounced as to render the victim less and less capable of exertion. In spite of this condition, however, the general health may be but little impaired, and unless the dyspnea be too great, the patient may not be compelled to cease work. As time goes on the sputum which at first is mucoid in character, becomes mucopurulent and blood-streaked or even a frank hemoptysis may occur.

Even when the fibroid changes have become marked, the physical signs may indicate nothing more than a diffuse bronchitis. There is quite likely to be added to the picture, however, evidences of bronchiectasis. Marked clubbing of the fingers and the expectoration of large quantities of sputum, sometimes of a foul odor, are strongly suggestive of an associated dilatation of the bronchi.

If the condition becomes complicated with tuberculosis, there is little to distinguish the two conditions so far as the physical signs are concerned. The constitutional symptoms, however, may become marked: there may be fever, a rapid pulse, night sweats, emaciation, and most important of all, tubercle bacilli are present in the sputum.

The distinguishing feature of general fibrosis of the lungs is the contrast between the extensive pulmonary lesions and the absence of marked constitutional symptoms. "Herein fibroid phthisis presents a well-marked difference from pulmonary tuberculosis; and even if, as we have said, the disease becomes complicated with tubercle, yet the rate of progress may be determined rather by the character of the primary than of the secondary disease, though usually the supervention of tubercle hastens the sufferer into a more rapid consumption."[1]

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[1] Report of British Departmental Committee on Compensation for Industrial Diseases, 1906, p. 13.

Physical Signs.—The physical findings in cases of pneumoconiosis e most varied, depending, as they do, upon the stage of the disease d whether it is complicated or not. In the early stages of the process amination of chest may reveal nothing unusual, or, at most, the signs dicative of a bronchitis. As the fibrosis increases, however, abnoralities are more and more frequently found.

Inspection.—When the disease becomes fully established it will be ted on inspection that expansion of the lungs is deficient; usually this more marked on one side. If the disease is more marked on one side an the other, there is also apt to be some retraction of the chest wall. vidences of poor expansion and retraction may be present at the apices at the bases, usually the latter. When the left lung is involved, e heart is drawn to that side and, in addition, the area of cardiac pulse may be increased by reason of the retraction of the lung. Ex- nsive fibrosis of the right lung will cause the heart to be drawn to the ght. Distention of the veins in the neck may occur as the result of latation of the right heart.

The chest may be of the emphysematous type but as a rule the em- ysema is not sufficiently marked to bring about this change.

As the result of an associated dilatation of the bronchi the fingers may markedly clubbed.

Palpation.—This will aid in determining the areas of deficient expan- on, the location of the apex beat of the heart and the portions of the ngs which have become solidified.

Percussion.—In the beginning the percussion note is normal. If nphysema plays a prominent part the note will be hyperresonant. In e majority of instances the percussion note becomes impaired or nearly ll in certain areas, namely, the apices, the bases or the interscapular gions. The dull areas may be bilateral or unilateral. If bronchiectasis pervenes, a tympanitic note may be obtained. The common sites for ting this change are either beneath the clavicle or near the angle of the apula. Owing to fixation, partial or complete, of the diaphragm the spiratory excursion of the lungs at the base is very slight or absent.

Auscultation.—The auscultatory signs are numerous and varied. the formative stages of pneumoconiosis nothing abnormal is detected. s the exposure to the dust becomes more prolonged râles, both large and nall, may be heard in both lungs. At this time the breath sounds may semble those encountered in emphysema, namely, a rather feeble spiratory murmur with prolongation of expiration.

When the fibrosis becomes at all marked there are apt to be certain reas over which the breathing is suppressed and broncho-vesicular. Fine rackling râles are also present at these points and the voice sounds are xaggerated. Attacks of pleurisy are not uncommon so that a friction ub may be noted. When the fibrosis is diffuse and there are no localizing igns, the breath sounds over the lungs may be slightly harsh and roncho-vesicular in character. Râles are usually constantly present ut at times are more marked than at others as intercurrent "colds" re quite common.

If the dilatation of the bronchi becomes at all marked, cavity signs whispering pectoriloquy, cavernous breathing, metallic or resonating âles) are obtained. The cavity signs may be detected at the apices or he bases of the lungs. When heard about the angle of the scapula, they are always suggestive of bronchiectasis; at the apices they may be due to an excavation, the result of tuberculosis or dilated bronchi.

The heart, as a rule, presents nothing characteristic of pneumoconiosis.

X-ray Examination.—The study of this condition by means of the roentgen rays is the greatest advance which has yet been made. In the past few years several excellent reports of this nature have been published. During the past two years Pancoast, Miller and I[1] have studied several hundred individuals who have been exposed to dusts of various sorts.

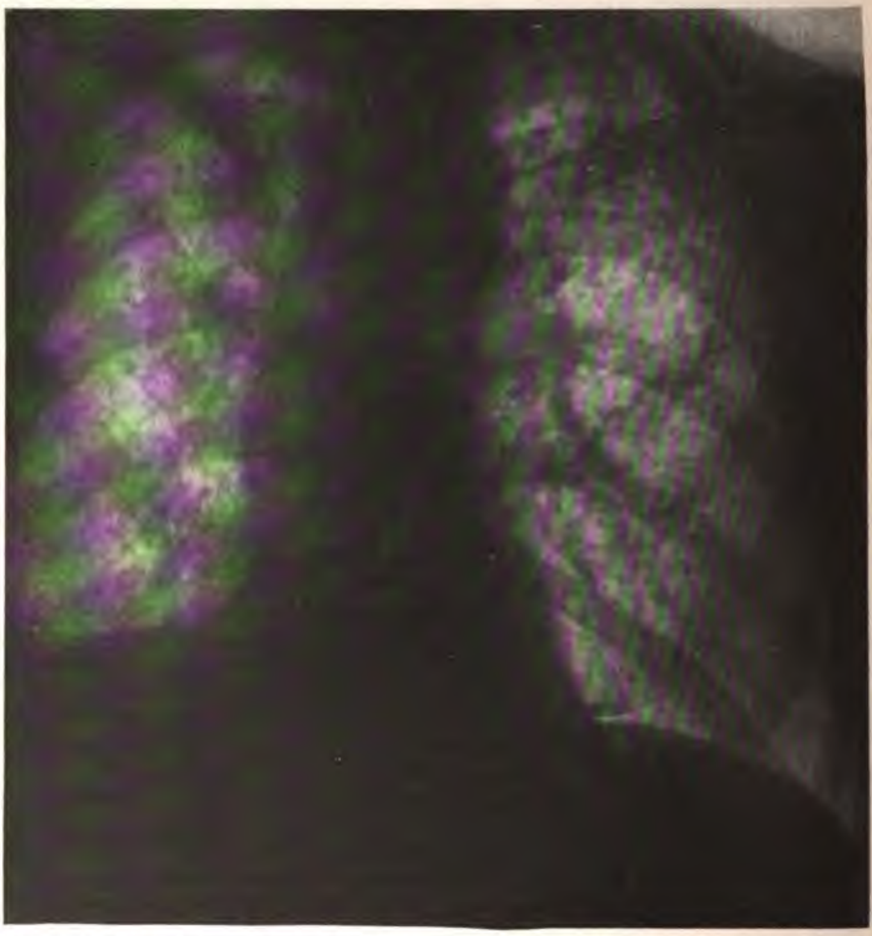

FIG. 306.—Second stage of pneumoconiosis with beginning fibrosis. Taken from a potter.

In regard to the effects of *organic dust* the subject can be dismissed with the statement that these individuals show no more marked changes than are encountered in those who have dwelt all their lives in large cities. When the evidence of pneumoconiosis is slightly more marked than normal in these cases, it is to be ascribed to the fact that there is a conderable amount of inorganic material mixed with the organic dust. his was notably so in a number of carpet makers we examined.

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[1] *Trans. Assoc. Amer. Phys.*, 1917.

The roentgenologic picture in the case of those exposed to *inorganic* st is, in the great majority of cases, characteristic except perhaps in e very early stage. As a rule, the changes as seen in the roentgeno- am correspond closely to the pathological processes.

The first stage is characterized by an increase in the hilus shadows and thickening of the usually prominent trunk shadows, and an undue ominence of the finer linear markings. It is the rule that the increase thickness of the bronchial trunk shadows is fairly uniform, which is e main dependence in distinguishing the case from one of peribronchial berculosis, but there are exceptions which make the roentgenologic gnosis difficult or uncertain. The descending trunk shadows seem be more marked on the right than the left but the interference offered the heart shadow on the left side may hide a similar condition there. normalities of the diaphragm excursion occur in all stages, even the st. In the first and second stages, however, there is no general rule. e position of the excursion during ordinary respiration between the nts of extreme inspiration and expiration is very variable. The most mmon finding, in our experience, was a restriction at the inner portion the right leaflet during full inspiration and either a decided flattening a concavity of the outer aspect.

The *second stage* is characterized by a more or less uniformly arranged ttling throughout the lung structure due to the deposition of dust the lymph spaces, cells and fibrous tissue interspaces, with the addi- n of a certain amount of localized fibrosis (see Fig. 306). This stage prises what has usually been regarded as the typical case of pneumo- iosis. Its onset seems to depend largely upon the character of the t inhaled. It occurs early in those exposed to silica; comparatively ly in coal miners, certain metal grinders and somewhat later in potters asbestos workers. The distribution of the mottling was found by to invariably appear first on the right side on a level with the hilus dow. It certainly becomes quite perceptible on the right side before appears on the left side and for some time after it does appear on the t side, it is noticeably more marked on the right. In the advanced ge the two sides seem to be about equally involved. The distribution more or less symmetrical, but naturally not uniform throughout the ng. From the starting point it gradually spreads to the bases and pex, but is never so marked at the extreme apex or base as around the d-portion of the lung. The appearance of the mottling depends more r less upon the character of the dust. In those exposed to silica the pots are very dense and sharply circumscribed and can be seen when ery small. In the case of the less irritating forms of dust the spots are ot so sharply defined. The roentgenologic diagnosis in this stage is ot difficult. It may be difficult, however, to detect an early tuberculous esion engrafted upon a well-advanced case of pneumoconiosis in the econd stage.

The *third stage* is characterized by the appearance of diffuse fibrosis nd all that the term implies. While there is no sharp dividing line etween the second and third stages, it would appear in some instances hat the mottled appearance in the second stage becomes more and more onglomerate and finally passes over into the appearance of dense fibrosis.

In other instances a general haze seems to spread over a certain portion of the lung. The greatest density is in the subapical regions, although this is not the area of most intense mottling in the second stage. Dense fibrous bands can be seen extending in various directions, and frequently to the diaphragm, causing marked retraction (see Fig. 307). In this stage the mottling has become extremely coarse and sometimes is to be no longer recognized as such. The heart and blood-vessels are frequently dragged out of place and bronchiectatic cavities are quite common.

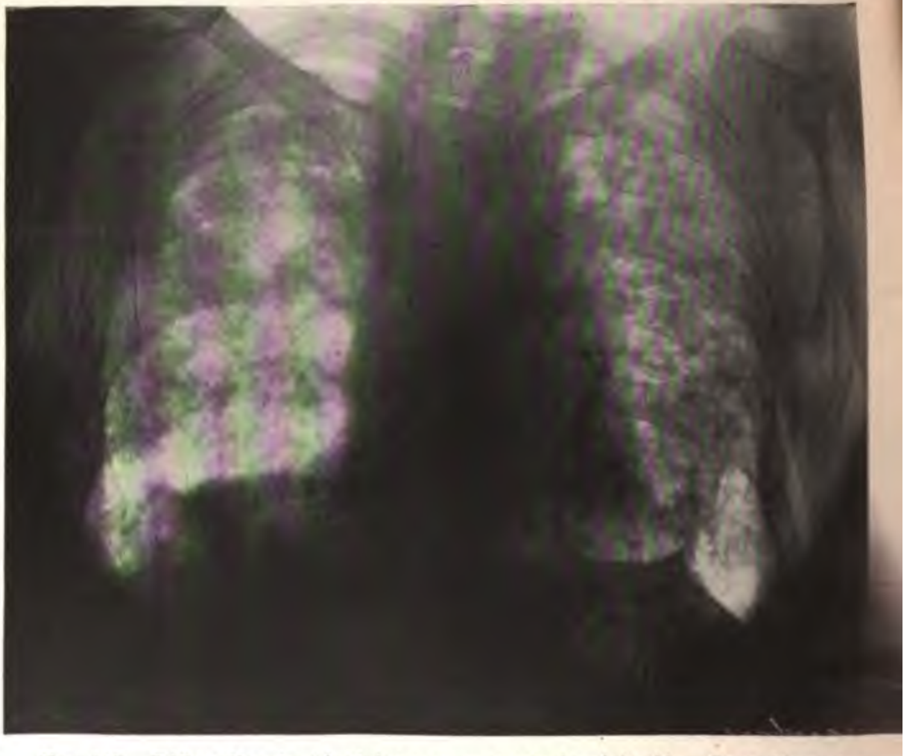

FIG. 307.—Diffuse fibrosis of both lungs, more marked on left side. Bronchiectasis at both apices and some emphysema at right base. Note also the angulation formed in the left leaflet of the diaphragm. From a male, age 55, employed as a potter for 45 years. Exposed to clay dust. Principal symptoms: cough, expectoration and dyspnœa. Sputum examinations repeatedly negative for tubercule bacilli. Physical signs identical with those encountered in advanced tuberculosis. (*Radiographed by Dr. H. K. Pancoast.*)

It is not always easy to differentiate between the fibrosis resulting from tuberculosis and pneumoconiosis and were it not for repeated negative sputum examinations, sometimes extending over a number of years, it would not be easy to say that tuberculosis was not present.

Diagnosis.—The diagnosis of this condition in the first and second stage is possible only by means of the roentgen rays. Its presence and the stage of its development can be fairly accurately summarized, however, if one knows the character of dust to which the individual has been exposed and the length of the exposure. As a general rule an individual suffering from pathological changes in the lungs as the result of exposure to dust almost invariably seeks relief because of shortness of breath, cough and expectoration, or a gradual failure in strength. The clinical picture is that of chronic bronchitis, emphysema and asthma, bronchiectasis or last, but most important, tuberculosis. The recognition of pneumoconiosis depends, therefore, on a knowledge of the occupation of the patient. If the history indicates that there has been prolonged exposure to an irritating form of inorganic dust it is almost certain that the symptoms and physical signs no matter how slight or how varied they may be, have had their origin in this way. A knowledge of the rôle played by dust is important, because the process in the lung can be arrested if the individual seeks a dust-free occupation.

Pure pneumoconiosis can be distinguished from that with a complicating tuberculosis, only by means of sputum examinations.

ATELECTASIS

By atelectasis (ἀτελης, incomplete; ἐχτάσις, expansion) is understood the return of lung tissue, once expanded, to a retracted, airless state. The terms collapse of the lungs, état fetal, and apneumatosis are sometimes employed to convey the same meaning.

Etiology.—Atelectasis may be (1) congenital or (2) acquired.

1. Up to the moment of birth the lungs are solid organs, the alveolar walls lying in close contact. With the first inspiration the lungs undergo partial expansion, the alveolar walls being separated and the bronchioles assume their permanent shape. If the child has not breathed at all, the lungs present throughout the color and texture of the adult liver. If a few respirations have occurred, the surface of the lung is studded with distended air vesicles while the remainder presents the appearance of atelectasis. Congenital atelectasis is met with in still-born children at term, in prematurely born children and is often seen in those with congenital syphilis, lesions of the central nervous system or as the result of obstruction of the bronchi by foreign bodies or secretions. In weakly children it is probable that portions of the lungs remain unexpanded during the first year of life.

Congenital atelectasis is chiefly of medico-legal interest.

2. Acquired atelectasis may arise as the result of (*a*) interference with the respiratory function within the lung itself and (*b*) external mechanical pressure.

(*a*) *Interference with the respiratory function* may be brought about, by obstruction in the upper air passages due to enlarged tonsils, adenoids or laryngeal stenosis.

Another cause of atelectasis is obstruction of the bronchi. This may be brought about by inflammatory swelling of the bronchial mucous membrane and the accumulation of secretions within the tubes in such conditions as bronchitis, broncho-pneumonia and whooping cough. The bronchi may be occluded also as the result of a foreign body or by pressure from without by an aneurism or mediastinal growth.

Finally a very common form of atelectasis, due to interference with the respiratory function, is met with in cachectic or moribund individuals. In such cases the weakening of certain groups of muscles concerned in respiration fails to support the chest wall. As a result the cavity of the thorax which should be enlarged by the descent of the diaphragm becomes narrowed and the intercostal spaces and lower parts of the chest recede during inspiration thus causing the underlying parts of the lungs to collapse. In conditions characterized by extreme weakness whether from old age, prolonged fever, or other cause, small areas, partially or completely collapsed, chiefly at the margins and bases of the lungs, are of frequent occurrence.

A long continuance of the above causes, especially when due to enlarged tonsils, adenoids or weak muscles, may lead to permanent deformity of the chest such as the "pigeon breast" or the development of marked lateral depressions (Harrison's furrow).

(*b*) *The lung may be compressed by external pressure.* The pressure may be brought about as the result of a pleural effusion, pneumothorax (spontaneous or artificial), aneurism, tumor, cardiac hypertrophy, pericardial effusion, thickening and contraction of the pleura and by spinal deformities (see Figs. 103, 105, 107, 109). All of these causes operate not only by direct pressure but also by interfering with the proper respiratory movements. Pasteur[1] has drawn attention to collapse of one of the lower lobes due to reflex inhibition of the diaphragm. This condition is sometimes encountered after abdominal operations.

Morbid Anatomy.—Atelectasis may affect one or both lungs. It may be limited to small circumscribed areas or it may involve an entire lung or any portion thereof. When occurring in the circumscribed form, the collapse areas are usually to be found in those parts of the lung where the expansile power of the chest is least, and which are the earliest to be affected by muscular weakness. Hence, atelectasis is most commonly found at the bases and along the free margins of the lungs. The middle lobe of the right lung is frequently involved. In the lobular form, if situated at the periphery of the lung, the involved area is sharply defined, shrunken, depressed below the surface and of a dark violet tint. On section it is smooth and glistening in appearance and on pressure a small amount of serum exudes. The tissue not only feels airless but also sinks when placed in water. In the early stages the surrounding lung tissue may be edematous; later it usually undergoes emphysematous changes. If not of long standing, the collapsed cells may become reinflated. If, however, they remain permanently collapsed, interstitial changes usually take place.

When the lung as a whole becomes collapsed from pressure without, as in the case of a pleural effusion, pneumothorax, etc., the organ is greatly reduced in size and lies high up in the thoracic cavity close to the spinal column (see Fig. 344). The lung has a leathery feel, the surface is thrown into fine wrinkles and is of a purplish or brownish-red color. If the pressure is marked, the organ becomes ischemic and of a grayish or slaty color. On section the tissue resembles flesh, does not crepitate and sinks in water. Providing the pressure has not existed for too long a time the lung will gradually reëxpand. If, however, the collapse has been of long standing the lung gradually undergoes interstitial changes and finally is transformed into a fibrous mass with or without dilatation of the bronchi. In the early stages of collapse the interference with the blood supply renders the lung susceptible to infections such as bronchitis or broncho-pneumonia.

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[1] Annual Oration, Med. Soc., London, May, 1911.

Massive collapse of one of the lower lobes as a sequel to abdominal operations is due to paralysis of the diaphragm (see p. 633). Pasteur states that the deflation may be so complete that the affected portion sinks in water. In most instances this form of collapse is of brief duration, the lung becoming reëxpanded in a day or so.

Symptoms.—Pulmonary atelectasis may occur without any clinical manifestations whatever. When present, symptoms indicative of pulmonary collapse are extremely variable and depend in great measure on its extent and on the acuteness and rapidity of its production. Thus collapse of the lung may be attended by sudden and alarming symptoms as in cases of spontaneous pneumothorax or it may show itself only by slight shortness of breath on exertion as in cases of pleural effusion. In the great majority of instances in which pulmonary atelectasis is present the phenomena produced by this condition form a part of the clinical picture of other well-recognized disorders.

Complete bilateral atelectasis is seen in still-born children only. The condition is of great medico-legal interest as it proves that death occurred prior to birth.

In prematurely born children and in those enfeebled by some constitutional taint such as syphilis, the lungs may become partially expanded. In such children the temperature is low, and they do not become red or pink, but retain a blue color. They are often called "blue babies" although this term should be reserved for infants born with heart disease. The respirations are rapid and superficial. The face is pallid and pinched, the lips and hands cyanosed and the eyes and fontanelles are sunken. General weakness is very marked and the child has difficulty in suckling. Furthermore it does not cry with vigor, but whines, and each expiration is attended by a grunt or a light groan. As a rule, this condition lasts for a few days when the cyanosis becomes more marked, the child lies in a somnolent state and finally develops twitching or convulsive movements of the extremities. While the duration of life in these children is usually but a few days, death may not occur for several weeks. If the cyanosis is marked, the child rarely lives more than a few days.

When collapse of portions of the lungs occurs as a complication of bronchitis, broncho-pneumonia, whooping cough or any condition attended by blocking of the smaller bronchi, its presence is to be inferred largely by an increase in the severity of symptoms. As a complication of broncho-pneumonia the onset may be sudden and severe. There is an urgent sense of dyspnea and respiratory distress, the respiratory and pulse rates become rapid, the movement of the alæ nasi exaggerated, and the child becomes restless and fretful. The cough may be ineffectual due to imperfect inspiratory expansion or it may be violent and paroxysmal in character. Gradually the child becomes more and more cyanosed, sweats profusely and sinks into unconsciousness or dies during or after a paroxysm of coughing.

Pulmonary collapse which occurs as the result of pressure exerted by a pleural effusion, tumor, aneurism, etc., is rarely attended by marked symptoms. As a rule the condition has been brought about so gradually that the opposite lung has had time to accommodate itself to the change. If, however, a pleural effusion develops rapidly or entirely fills one side of the chest, there is dyspnea, especially on exertion, and a sense of oppression.

The most obvious form of atelectasis is that which occurs as the result of pneumothorax. While it is true that pneumothorax sometimes develops gradually and produces no symptoms the majority of cases are characterized by a sudden onset. In such instances the lung collapses so suddenly that the intact lung has thrust upon it, without a moment's warning, the entire burden of the respiratory function. The sudden displacement of the heart and great vessels and the disturbance of the intrathoracic pressure still further aggravates the situation. Sudden collapse of an entire lung is attended by all the phenomena of shock, namely, pallor, a clammy skin, marked anxiety and a rapid, feeble pulse. In addition there is air hunger and the breathing is hurried and tumultuous. Death may occur instantly, within a few hours or recovery from the attack may gradually take place. If the opposite lung is healthy or but little diseased, it is able to accommodate itself to the situation and recovery usually occurs. Fatal cases occur, as a rule, in tuberculous individuals in whom the opposite lung is also diseased.

Collapse of the lung is also brought about artificially by the introduction of nitrogen gas into the pleural cavity. Aside from some slight difficulty in breathing and a sense of tightness in the chest lasting for a few hours, the compression rarely produces any disturbance. Occasionally death occurs suddenly during or just after the injection. The cause of the accident is not known.

Massive collapse of one of the lower lobes due to paralysis of the diaphragm sometimes develops with such suddenness and intensity as to suggest a pulmonary embolism. In other instances the condition gives rise to so few symptoms that the presence may be overlooked. The condition is as a rule transient in character. The dyspnea rarely lasts more than 24 hours. In addition to the dyspnea there may be a viscid expectoration. This is believed to be due to the contents of the collapsed lobe which are pressed into the bronchi and coughed up.

Physical Signs.—Because of the diversified conditions under which atelectasis occurs, a detailed description of the physical signs is not feasible. In some instances the condition does not give rise to distinctive signs and its existence must be a matter of inference, as for example in broncho-pneumonia. In those cases in which the entire lung is partially or completely collapsed as the result of mechanical pressure the physical signs are to be attributed to the causative agent such as a pleural effusion or air in the pleural cavity. The physical signs obtained over the lung after absorption or withdrawal of fluid have been dealt with under the heading, "Pleural Effusion."

In prematurely born or asthenic children the intercostal spaces and the lateral and inframammary regions of the chest will be seen to recede during inspiration. The percussion note over the bases of the lungs is usually impaired and the respiratory murmur everywhere weak. Inspiratory recession of the interspaces and lower parts of the chest may also occur in cases of collapse associated with broncho-pneumonia and in cachectic and weakened individuals. In the latter, fine crepitant râles may be heard as the result of an associated edema.

Collapse of a lobe or portion thereof shows absence of expansion, some retraction of the chest wall, dulness on percussion and absent or int bronchial breath sounds. If edema is superadded to the collapse, ne crepitant râles are also heard. Pasteur emphasizes the importance f noting the position of the apex beat of the heart in cases of collapse of ne of the lower lobes. The displacement of the apex beat is always ward the affected side.

Diagnosis.—When the collapse occurs suddenly and involves a large ortion of one lung the condition may be mistaken for croupous pneu- onia. The absence of inflammatory symptoms and low temperature ogether with retraction of the chest wall and displacement of the apex eat of the heart serve to distinguish the two conditions. Collapse of a rge part of a lung sometimes occurs in children from slight causes, pecially catarrhal conditions. Hence when the signs of consolidation re found in a young child, we must always remember that they are pos- bly due to atelectasis (Gee).

Considerable difficulty is experienced in distinguishing between ollapse of a lower lobe and the presence of fluid in the pleural cavity ven when fluid has previously been withdrawn. In both conditions here is absent or distant breath sounds, dulness on percussion and absent xpansion. If the case has been followed from the beginning the gradual eturn of the breath sounds can be noted but when seen for the first time t may be necessary to introduce an exploring needle to settle the question.

EMPHYSEMA

By pulmonary emphysema is meant an abnormal inflation and ilatation of the air cells, which may be temporary, or as is usually the ase, permanent in character. Five varieties are recognized; chronic ypertrophic; atrophic or senile; acute vesicular; interstitial; and com- pensatory.

CHRONIC HYPERTROPHIC EMPHYSEMA

The term emphysema, as usually employed, has reference to the hronic hypertrophic form which in most instances is a secondary process, he result of some preëxisting morbid process in the lungs or bronchi.

Etiology.—Chronic emphysema is rarely primary in character. The ssential feature of the condition is the loss of elasticity in the lungs. This may be brought about by any affection which causes a forcible and more or less continuous overdistention of the pulmonary tissue.

Strümpell illustrates this very clearly by comparing the sound lung with its normal elastic force, to a new and very tense rubber band, while the emphysematous lung may be compared to an old and lax band that is overstretched and pulled out. This loss of elasticity with the resultant inability of the lung to contract is seen when the thorax of an individual, subject to the disease, is opened. Instead of collapsing as does the normal lung under such circumstances, the emphysematous lung, being in a permanent state of inspiratory inflation, remains distended even after removal from the chest.

The expiratory theory of emphysema was first advanced by Mendelssohn and later, and independently, by Sir William Jenner. The latter expressed his views as follows: "The lung during expiration is compressed at different parts with different degrees of force. The parietes of the thorax, in consequence of their anatomical constitution, yield to the same force at different parts with various degrees of facility. The chosen seats of emphysema are exactly those parts of the lung which are the least compressed during expiration, and which are situated under those portions of the thoracic parietes that give way the most readily before pressure."

More recently Adami and Nicholls have explained the expiratory theory as follows: Take, for instance, the simplest example a localized emphysema in which the change affects one small bronchial tree and the associated air sacs, the rest of the lung being in a normal state. The primary difficulty is in the bronchus and this may consist of a narrowing of the lumen of the bronchus, whether congenital or induced by inflammatory thickening, or by the presence of thick exudate within it. In such a case the forcible nature of the inspiratory act draws air into the air sacs while the passive nature of the expiratory act may prevent an equal amount of air becoming expired. The result will be that with the successive acts of inspiration, the air sacs will become more and more distended, just as occurs in bronchial asthma as the result of spasm of the bronchioles. If this overdistention of the alveolar wall be continued, the result will be malnutrition due to interference with the capillary circulation and this in turn leads to atrophy and loss of elasticity.

Ever since the researches of Louis and James Jackson, Jr., the *hereditary element*, as an underlying factor in the causation of permanent dilatation of the air cells, has been accepted. The exact nature of this inherited tissue defect has never been satisfactorily explained. It is assumed, however, that in families subject to this affection there is a quantitative or a qualitative lack of pulmonary elastic tissue, and because of this such individuals become emphysematous much more readily than others. Emphysema developing before the middle period of life, and particularly when it manifests itself in youth, is probably the result of a congenital defect in the elastic elements of the lung. It is also to be noted that such individuals not infrequently are subject to hay fever and asthma.

Whether we are willing to accept the hereditary theory or not it is certain that the elastic tissues of the body tend to lose their elasticity with advancing years. This is true in regard to the arteries; that the same thing is true in regard to the lungs is evident from the fact that marked emphysematous changes are far more often encountered in those of advanced years than in younger individuals. Furthermore, the association of chronic emphysema and arteriosclerosis is a very frequent one.

The influence of *occupation* in the production of emphysema is a fairly well established fact. Under these circumstances the emphysema may be brought about in several different ways. Thus it may be produced by one who habitually handles heavy weights, by reason of the fact that heavy lifting is apt to be attended by rapid and deep inspirations, and also by increased pressure during expiration, owing to the closure of the glottis, which occurs in violent efforts. This forcible inflation and deflation of the lungs, if long continued, untimately wears out the elastic tissue. The playing of wind instruments, because of the prolonged overdistention of the lungs, is said to operate in much the same manner. Although cited as a frequent cause of chronic emphysema, we are of the belief that the importance of this particular vocation has been over-estimated.

From the standpoint of occupation, any employment which subjects the worker to the constant inhalation of fine *dust* particles must be considered as one of the most potent factors in the production of emphysema. In such cases it is almost invariably secondary to a chronic bronchitis. "Miner's asthma," while in some instances tuberculous in nature, is in many others a true hypertrophic emphysema having its origin in a hronic bronchitis, which in turn developed as the result of the constant nhalation of fine particles of coal dust.

Because of the nature of their employment, men are more subject o chronic emphysema than women. Extensive pleuritic adhesions by npeding the movements of the lungs may lead to diminution of the lastic force, probably as the result of increased expiratory pressure.

In the majority of instances, however, we must consider chronic ronchitis as the most frequent and the most important exciting factor. he overdistention under these circumstances is brought about as fol-ows: With each coughing attack the glottis is closed and the chest alls are strongly compressed by the thoracic muscles. Thus in the xpiratory effort to overcome the obstruction above (the closed glottis) he lung is overstretched, the overdistention being first manifested in hose parts least protected, namely, the apices and anterior margins, egions in which the emphysema is always most marked. Heavy lifting, s we have already pointed out, acts in much the same manner.,

As a further result of this increased intrathoracic pressure, the ternum is gradually pushed forward, also the cartilages, the ribs eventu-lly assuming a position approximating to that seen at the end of the nspiratory act. In other words the chest becomes barrel-shaped.

Finally, mention must be made of the most recent theory as to the ausation of emphysema, namely, premature ossification of the costal artilages. This explanation has been advanced by Freund, who is of the belief that the ossification of the costal cartilages is the cause of the rigid dilatation of the chest, and the associated emphysema rather than one of the results of the latter.

Although calcification of the costal cartilages is commonly present in cases of chronic emphysema, the theory advanced by Freund has received but little recognition.

Morbid Anatomy.—Chronic hypertrophic emphysema is a bilateral affection. While both lungs are involved more or less uniformly, the majority of cases show more marked evidences of inflation at the apices, he anterior borders and the inner surface near the root of the lung. The chest of an emphysematous individual is barrel-shaped, all the liameters being increased and particularly the antero-posterior. The ostal cartilages are usually lengthened and in addition may be calcified. On opening the chest the lungs are seen to be greatly inflated and cover he heart. They do not collapse but may even project on opening the hest. The lungs are pale, less crepitant than normally, are dry and give he sensation of handling a feather pillow (Fig. 308). Scattered over the leural surface may be seen small vesicles the size of a pinhead; in advanced cases the vesicles may attain the size of peas, giving the lung a ullous appearance. A spontaneous pneumothorax sometimes results rom rupture of one of these vesicles. On section the lung is seen to be lry and very little blood escapes. This is due to the fact that many of he capillaries in the alveolar walls have been destroyed through stretch-ng. The bronchi usually show evidences of a chronic bronchitis and some thickening of the bronchial wall; dilatation of the bronchi may be present also.

Microscopically the vesicles are seen to be distended and the walls thin and atrophic. As a result of the distention of the alveolar walls the capillary circulation is interfered with. The capillary vessels may be elongated, narrowed or entirely obliterated. The septa between adjacent alveoli become wasted and thin and as the disease progresses they rupture. In this way two or more alveolar spaces may be thrown into one. The blebs or vesicles seen beneath the pleura and along the fre margins are formed in this way.

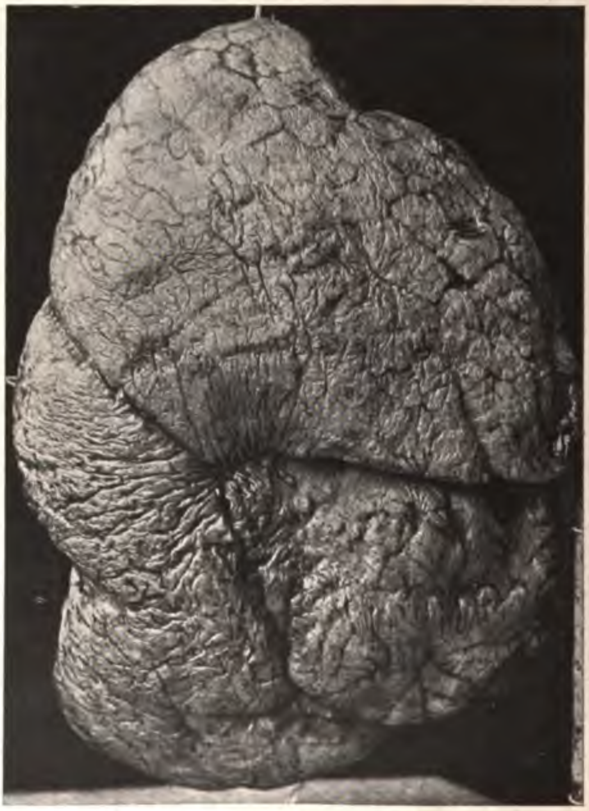


FIG. 308.—Chronic emphysema. Dry specimen.

The effect of a generalized chronic hypertrophic emphysema is t alter the contour of the chest and increase it in size; to bring about form of dyspnea characterized by difficulty in expelling the air from th lung; to obstruct the passage of the blood through the lungs with th result that hypertrophy, first of the right heart and later the left hear takes place and eventually cardiac failure. Interference with the pu monary circulation may lead also to atheroma of the pulmonary arter

Symptoms.—The chief symptoms of emphysema are dyspnea an cough and expectoration, the latter symptoms being due to freque ks of bronchitis which finally becomes chronic. The dyspnea is not ;o be marked unless attacks of asthma are associated; in most nces exertion causes more or less shortness of breath. The cough expectoration vary greatly. Emphysematous individuals may be ely free from these symptoms during the summer months or if they l the winters in a warm climate. During the winter months they are ially liable to bronchial attacks. The longer the disease has lasted nore likely is there to be associated with it a chronic bronchitis.

s the individual advances in years the circulatory disturbances ne more and more marked. Shortness of breath increases on exer- cyanosis is often noted and finally the picture is that of failing ensation.

'hysical Signs.—*Inspection.*—Emphysema of varying grades of ity may be encountered, but the well-developed case presents certain ıres which render it easy of recognition almost at a glance. The

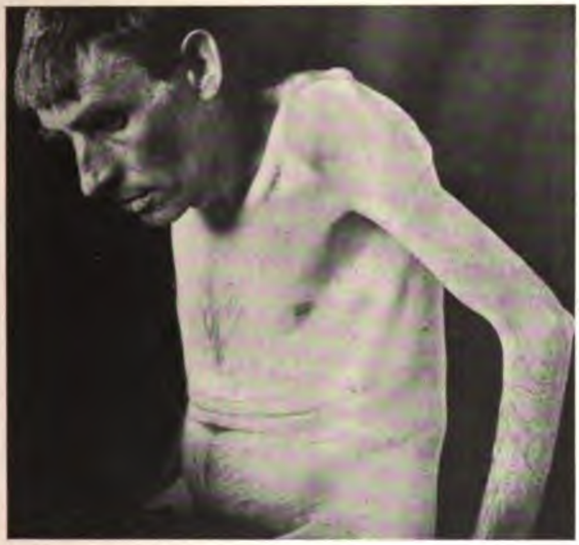


FIG. 309.—Chronic emphysema.

nt, while usually well nourished is seen to be somewhat cyanotic, times to a very marked degree; the veins of the neck are distended; sternocleido muscles are conspicuous and the neck seems shorter normal. The clavicles are prominent and the fossæ above them ually deep. The most characteristic feature, however, is the forma- of the chest, which is in a permanent inspiratory position, or as it been aptly termed, "barrel-shaped." The sternum projects, thus ng to increase the antero-posterior diameter of the chest, until it equal the length of the lateral diameter. The increase in the antero- al diameter is also augmented by the exaggeration of the normal l curve which gives the back a bowed appearance (Fig. 309). The ening of the chest tends also to shorten it from above downward the result that the ribs are in closer approximation. The lower ribs incline downward but slightly and may even become straight. (See Figs. 13, 25, 33.)

The chest of the typically emphysematous patient has very little lateral expansion, but moves up and down as a whole, especially in forced breathing.

The apex beat of the heart is not visible, partly by reason of some displacement downward and inward, and partly from being covered by the distended lung. For this reason marked epigastric pulsation is not uncommon in these cases.

Palpation.—Palpation reveals little of importance. Vocal fremitus is normal, or at most but slightly diminished.

Percussion.—The percussion note obtained over emphysematous lungs is more or less characteristic and has been variously designated as hyperresonant, vesiculo-tympanitic or "box-like" in quality.

Austin Flint taught that emphysema was always greater in the left upper lobe than elsewhere and that for this reason an error was sometimes committed in assuming that the less resonant note beneath the right clavicle was due to impairment, when in reality it was simply a diminution in intensity.

As has been pointed out, the heart is entirely, or in large part, covered by lung tissue. For this reason the area of cardiac dulness is absent or greatly reduced even when a considerable degree of cardiac hypertrophy is present.

Owing to the distention of the lungs, the area of pulmonary resonance is greatly increased downward, both anteriorly and posteriorly. The upper border of liver dulness is then lower than normal, and the resonance may extend, in extreme cases, as low as the costal margin. With failure of the right side of the heart, liver dulness may extend far below the costal margin as the result of hepatic engorgement.

Posteriorly the lungs may extend below the line of the tenth rib, which is the lower limit in a normal chest. The percussion note at the bases may be impaired if there is much secretion present in the lungs.

Auscultation.—In some instances, the only noticeable alteration in the breath sounds is a slight diminution in the intensity of the respiratory murmur, without other changes either as to quality or rhythm; or the respiratory murmur may be so feeble as to be inaudible. It is important to bear these facts in mind, as the commonly accepted teaching is that an alteration in the rhythm of the respiratory murmur is characteristic of the condition. The alteration in the rhythm consists in the inspiratory sound being comparatively short and the expiratory greatly prolonged so that the ratio of the former to the latter sound may be changed from 3 to 1 to 1 to 4. In some instances the inspiratory sound may be entirely wanting, the expiratory sound alone being heard. The expiratory sound is low in pitch and non-tubular in quality, thus differing from the prolonged tubular and high-pitched expiratory sound heard over consolidation.

Vocal fremitus is normal or but slightly diminished.

The presence of râles is dependent on the coexistence of chronic bronchitis or asthma or both. If the former, the râles are large and small, the latter being especially noticeable at the bases. If the patient is in addition asthmatic, auscultation will reveal from time to time large sonorous and piping sibilant râles. These râles may be so numerous as ɔmpletely obscure the breath sounds and may even be heard at some ınce from the patient. The heart sounds are somewhat muffled ow- o the intervening lung tissue. The second pulmonic sound is accen- ed as the result of stasis in the pulmonary circulation. In a certain ortion of cases a functional murmur is heard at the apex. With the ually increasing embarrassment of the right heart the cardiac omena become more and more marked, but differ in no particular failing compensation due to other causes.

SENILE EMPHYSEMA

(Small Lunged Emphysema; Senile Atrophy of the Lungs)

ı the strict sense of the word this is not a true emphysema at all. ıe name indicates, it is encountered in those of advanced years, and t one of the atrophic changes, which are commonly met with in the

Iorbid Anatomy.—When the chest is opened the lungs readily pse. They are small, dry, easily compressible and as a rule deeply ented. The heart instead of being completely hidden, as in the case ypertrophic emphysema, is more exposed than is normal. The les are usually enlarged as the result of wasting of the septa. Large ∍ are not common, but are occasionally encountered along the free ;in of the lung. Owing to the fact that a catarrhal inflammation of ıronchial mucous membrane is almost always present, the bronchial are thin, and at times slightly dilated.

ymptoms.—There are no symptoms distinctive of senile emphy- , but individuals so affected have usually suffered from a winter h and shortness of breath for years. If the bronchi become suffi- ly dilated the expectoration, several times a day, of large quantities ıutum may occur. It is advisable to examine the sputum of all patients, as a very latent and chronic tuberculosis may present exactly ame picture.

hysical Signs.—*Inspection.*—Individuals subject to this condition ıvariably of advanced years and present a shrivelled, withered-up ap- ınce. The thorax is rigid. The lower ribs are very obliquely placed, may be in actual contact. The expansion movements of the chest nuch less than normal.

'here is no displacement of the cardiac impulse.

alpation.—Vocal fremitus is normal, or at least but little diminished.

ercussion.—The percussion note is hyperresonant with an under- tympanitic quality, partly the result of the rigid chest wall, and y the result of the large air vesicles and dilated bronchi. The rficial area of cardiac dulness is normal, or slightly increased, be- ∍ of the atrophied lungs.

uscultation.—The vesicular murmur may be feeble and the expiration ɔnged; on the other hand, it may be practically normal. Râles, large and small, are present especially in the winter months.

ACUTE VESICULAR EMPHYSEMA

'his condition is a dilatation rather than an emphysema of the air there being no structural change in the alveolar cells. It is encoun- in individuals who have died from acute bronchitis involving the smaller tubes, or in asphyxia, if accompanied by violent inspiratory efforts. It is also encountered in any condition which obstructs the air from entering portions of the lung, thus throwing a strain on the alveoli in other parts.

The percussion note may be hyperresonant and the area of pulmonary resonance increased. High-pitched piping râles are usually present.

INTERSTITIAL EMPHYSEMA

This form is characterized by the formation of large bullæ in the interlobular and subpleural tissues. Rupture of one of these large bullæ near the root of the lung may lead to the extravasation of air along the trachea, and thence into the subcutaneous tissues of the neck. Bullæ situated beneath the surface of the pleura may rupture and thus produce a pneumothorax. Aside from these two facts, this condition has no clinical interest. It cannot be recognized by physical signs, and during life its presence can only be surmised in the event of either of the accidents mentioned above.

COMPENSATORY EMPHYSEMA

(Vicarious or Complementary Emphysema; Hypertrophy of the Lung)

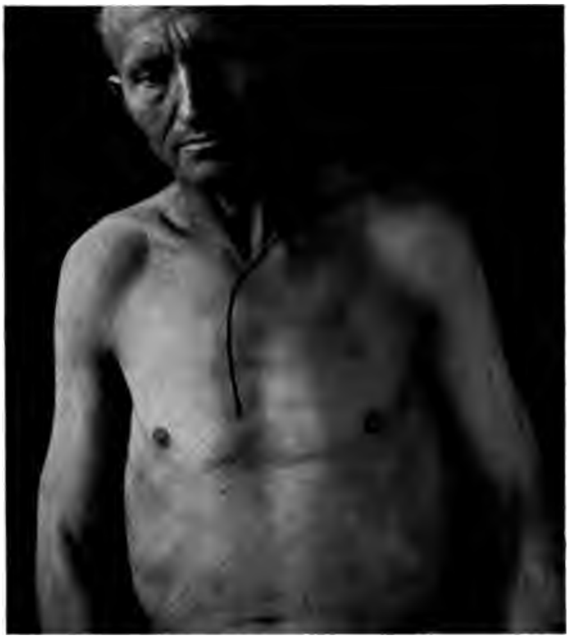
Compensatory emphysema differs from the forms considered above in that it is a *physiological and not a pathological process*. The condition may be localized in parts of one or both lungs, or it may involve one entire lung. Both the localized and the massive forms may be transient or permanent in character.

Any acute inflammatory affection of the lungs may be accompanied by a temporary form of emphysema. It is quite a common finding in the lungs of those who have died from broncho-pneumonia. In such cases the air vesicles between the areas of consolidation are somewhat larger than normal. This form is not recognizable clinically.

Localized emphysema, compensatory in nature, also occurs in certain chronic affections of the lungs, notably in tuberculosis of the apices, during both the active and healed stages of that disease. This is of considerable importance as the emphysema may mask the physical signs which otherwise would be detected over the infiltrated area in the incipient stage of tuberculosis. It may become permanent in character as one of the sequences of a healed tuberculous lesion.

The massive form is brought about by both acute and chronic pulmonary or pleural affections. In its temporary form it is most frequently seen in association with a pleural effusion, as a result of which the lung on the affected side is entirely or in large part unable to functionate. In the case of a serous effusion, whether as the result of an inflammation of the pleura itself, or as a consequence of chronic cardiac disease, the vicarious duty of the opposite lung ceases with the withdrawal or absorption of the effusion, and the reëxpansion of the compressed lung. If, however, the effusion is purulent in character, and is not promptly drained, the lung on the affected side becomes firmly and permanently anchored in its compressed position. The compensatory emphysema then becomes permanent and constitutes a true hypertrophy of the lung. Extensive tuberculous disease of one lung, especially of the chronic type with extensive fibroid changes, also leads to a marked degree of compensatory hysema of the opposite side. And this is true of any chronic inflam- ɔry, disease affecting one lung alone. In other words the presence pathological lesion involving all or a considerable portion of one lung ractically always accompanied by a compensatory emphysema of ɔpposite side.

'hysical Signs.—In some instances the hypertrophy of the unaffected is extraordinarily great. This may be apparent on inspection alone. sound side is seen to be much larger, and the range of the expansile ements unusually large. This is more apparent because of the nt or very restricted motion of the opposite or diseased side. Further- ɔ, the latter side is not uncommonly retracted. If the right lung ypertrophied, the apex beat of the heart will be seen to have been laced to the left, partly because of the mediastinal contents being



310.—Chronic fibroid tuberculosis of right lung. Compensatory emphysema of left lung which extends to the right border of the sternum.

rded over to the left by the enlarged lung, and partly because it is ed over by fibroid changes on the left side. In some instances with t-sided hypertrophy and left-sided disease, the heart is drawn up- l, the apex beat being most marked in the fourth or even the third rspace. If the left lung is hypertrophied and the right diseased, heart is displaced to the right in exactly the same way. In both ınces the extent of the hypertrophy on the one hand, and the disease he other will determine how great the displacement will be. It is uncommon to see the heart partly pushed and partly pulled so far to right as to resemble a case of dextro-cardia.

[he displacement of the heart *toward* the diseased side instead of y from it, distinguishes this condition at once from a pleural effusion.

Palpation confirms most of the above findings. (In addition there may be tactile changes over the diseased side; but the changes are apt to be variable, depending to a great extent on the degree of the fibroid changes in the lung and pleura.) Over the hypertrophied side the tactile phenomena are usually normal, or but slightly increased.

Percussion.—It should be recalled that the anterior edges of the lungs do not extend to the middle line anteriorly, and that posteriorly the bases are on a level with the tenth dorsal vertebra. (See Figs. 67 and 68.) With hypertrophy of one lung the anterior border will be found beyond the mid-sternal line, and in extreme cases may extend as much as 2 or 3 inches into the opposite side (Fig. 310). Posteriorly the resonance may extend from ½ to 2 inches below the normal limit. The anterior border and base of the affected side, on the other hand, are retracted. In tuberculosis it sometimes happens that the hypertrophied or healthy lung extends so far into the diseased side that it becomes infected by direct contact. (See Fig. 183.) One instance is recalled in which a cavity located during life in the left lung was shown at the autopsy table to be in the anterior border of an enormously hypertrophied right lung, which extended far over into the left chest.

Auscultation shows no abnormalities other than the exaggerated character of the vesicular murmur. This is very loud and is more harsh than normal. It bears a very strong resemblance to the puerile type of breathing met with in young children. The vocal resonance is normal or but slightly increased.

PULMONARY ABSCESS

Abscess of the lung is usually secondary to some primary inflammation of the lung tissue; as the result of extension from an adjoining organ, such as cancer of the esophagus or abscess of the liver; and, finally, it may be one of the manifestations of a general pyemic process.

Abscess of the lung may be either single or multiple. Inasmuch as the exciting causes, the symptoms and the physical signs of the two forms of abscess present, as a rule, certain marked differences, they will be described separately.

SINGLE ABSCESS

Etiology.—Local lesions within the chest play the predominant etiological rôle in the case of a single abscess. In a series of 30 cases of single pulmonary abscess reported by the authors[1] the exciting causes were as follows:

Croupous pneumonia	9
Vegetative endocarditis (infarct)	4
Suppuration in the abdomen	4
Broncho-pneumonia	2
Rupture of cancer of esophagus into the lung	1
Thrombosis of branch of pulmonary artery (typhoid)	1
General septicemia	1
Not determined	8
Total	30

The importance of croupous pneumonia is shown by the fact that the abscess followed that disease in one-third of the cases. In nearly another

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[1] Norris and Landis: *Trans. Assoc. Am. Phys.*, 1913.

'd of the cases it was not possible to determine the exciting cause. s quite possible that in the case of an apparently primary abscess, original source of the trouble was a latent and unrecognized pneumonia.

Within the past few years attention has been directed to the frequency h which a pulmonary abscess follows operations on the upper respira- r tract and particularly after tonsilectomy. Manges[1] and Claytor[2] 'e reported a number of cases. Of 26 cases of abscess which followed rations Wessler[3] states that 21 followed a tonsillectomy. The pulmo- y abscess may develop either as the result of a septic embolus, which ers the circulation at the seat of the operation, or by direct inspiration nfected material during the period of anesthesia. Caseous foci in the sils seem to be especially dangerous and for this reason the tonsils uld be removed with as little trauma as possible.

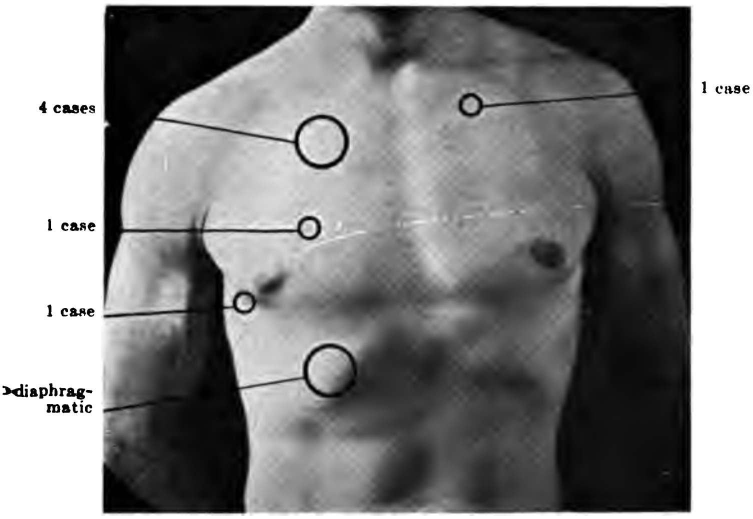

FIG. 311.—Location of abscess. Circles indicate site and size of abscess.

A pulmonary abscess may be one of the sequels of a traumatic injury he chest, such as violent contusion of the chest wall, a fractured rib, a penetrating gunshot or stab wound.

In addition to the above-mentioned causes of single abscess, I have l under observation two cases in which the abscess was caused by a ign body—in one case a tooth and in the other a brass-headed tack. e importance of foreign bodies as an etiological factor of pulmonary cess is, as the result of X-ray examinations, bronchoscopy, and of reasingly frequent operative intervention becoming more evident. a series of 25 cases Klübs[4] reports 7 as being caused by a foreign body d Guirez[5] has recorded 5.

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[1] *Amer. Jour. of Surgery,* March, 1916.
[2] *International Clinics,* Twenty-sixth Series, vol. ii, 1916.
[3] *Amer. Jour. Roentgenology,* April, 1919.
[4] "Ueber Lungenabszesse und Bronchiektasen," *Mitteilungen aus den Grenzgebieten. Medizin u. Chirurgie,* Heft 3, 549.
[5] *Bull. de la Socielie de Pediatric de Paris,* 1912, xiv, 56.

Morbid Anatomy.—In our series the abscess involved the right lung in 19 and the left in 11 cases. Figs. 311 and 312 show the site of the abscess. It will be noted that 21 of the 30 were located in the lower lobes. This is in accordance with the findings of others. Walker[1] in 132 cases col-lected from the literature, found the following distribution: upper lobes 21; lower lobes 76; middle lobe (right side) 2; both upper and lower lobes 5; in 28 instances the lobe was not indicated. Not only are the lower lobes the points of election but the right side is involved about three times as often as the left. This is doubtless due to the more fre-quent occurrence of croupous pneumonia in the right lower lobe. Wess (*loc cit.*) states that there is a distinct difference in the localization

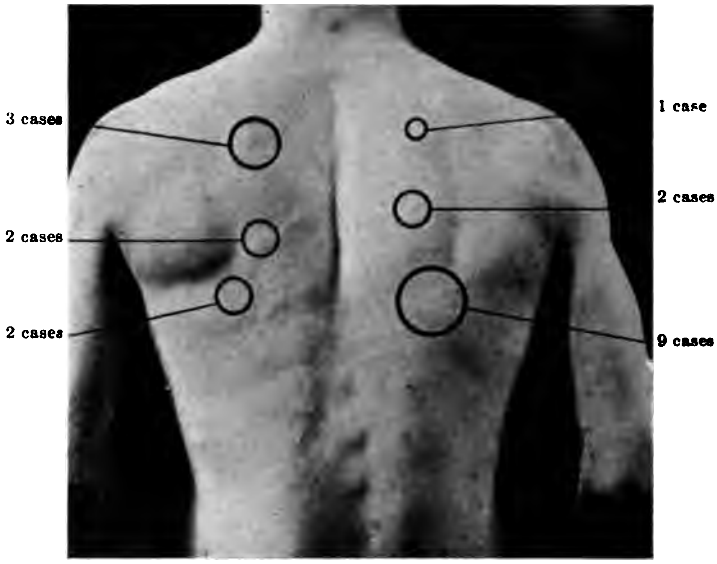

FIG. 312.—Location of abscess. Circles indicate site and size of abscess.

abscesses following the aspiration of infected material and those following pneumonia as will be seen from the following table:

	Upper lobe	Lower lobe	Middle lobe
Aspiration abscesses..............	18	9	1
Post-pneumonic abscesses.........	24	44	3

The size of the single abscess varies greatly, ranging from the size of a small marble to one with a diameter of from 10 to 12 cm. (Figs. 311 and 312).

A pulmonary abscess is usually round in shape. The involved area may be gray, yellow, reddish, or of a reddish-yellow color. On section it is composed of thick yellowish or whitish yellow or blood-stained pus. The pus may be odorless or it may have a sweetish or a very foul odor. If the abscess is recent and actively progressing, the wall is composed of soft necrotic lung tissue. Immediately surrounding this there is an area of intense hyperemia and edema. If the abscess is located near the

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[3] *Boston Med. and Surg Jour.*, July 9, 1914

ce, the overlying pleura will show some cloudiness and congestion.
abscess is quickly evacuated the necrotic wall of the cavity sloughs
nd is expectorated and the infection dies out. It is no longer
itant and the surrounding pulmonary inflammation subsides. The
may be completely obliterated, the only trace of its presence being
strands of fibrous tissue. Curiously enough a very large abscess
is as readily obliterated as a small one.

hen the abscess has existed for some time, connective tissue de-
s in the wall thus limiting and encapsulating the pus. The longer
scess remains the thicker and more dense the wall becomes. Fig.
ows a dense shadow in the right upper lobe due to a large abscess.
14 shows the same case after the abscess had spontaneously emptied

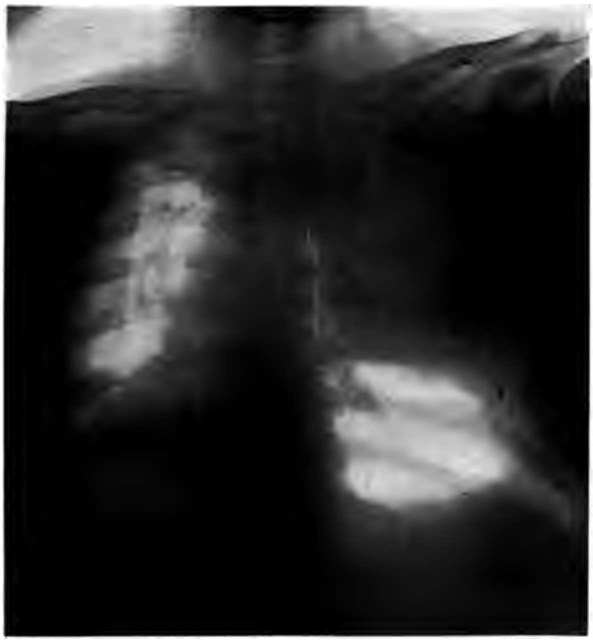

FIG. 313.—Pulmonary abscess in right upper lobe. (Posterior view.)

Occasionally instead of a well-defined abscess there is an area of
lly broken-down lung tissue infiltrated with pus and having the
rance of a sponge.

single abscess may be acute or chronic. In the chronic type the
ither becomes completely walled off or is not effectively drained.
her case it acts as a foreign body and the lung in the vicinity of the
s becomes fibroid. Even a relatively small abscess may lead to
sive fibroid changes with marked physical signs and symptoms. It
ly probable that many of the cases of chronic inflammatory changes
lower lobes, the etiology of which is uncertain, may have originated
s way.

single abscess may terminate in one of three ways: (1) It most fre-
ly ruptures into a bronchus and thus empties itself.

age is efficient, the process heals and unless the cavity is very large, nothing but a fibrous scar remains. (2) The abscess may rupture through the pleura. If this happens a pyo-pneumothorax sometimes results. The occurrence of this accident has been reported in a number of cases in the recent influenza epidemic. The more usual course, however, is the formation of an extrapulmonary abscess. Under these circumstances the pus may become encysted in an interlobar fissure, between the base of the lung and the diaphragm or between the chest wall and the lung. When an abscess forms near the surface of the lung, the pleura becomes inflamed and the two surfaces adhere. This acts as a defensive barrier if the pus breaks through the pleura and prevents the occurrence of a pyo-pneumothorax. Robinson is of the opinion that the majority of instances of encapsulated empyemata have their origin in a pulmonary abscess which ruptures through the pleura. (3) The abscess may become chronic and be the cause of extensive fibroid changes in the adjacent lung tissue.

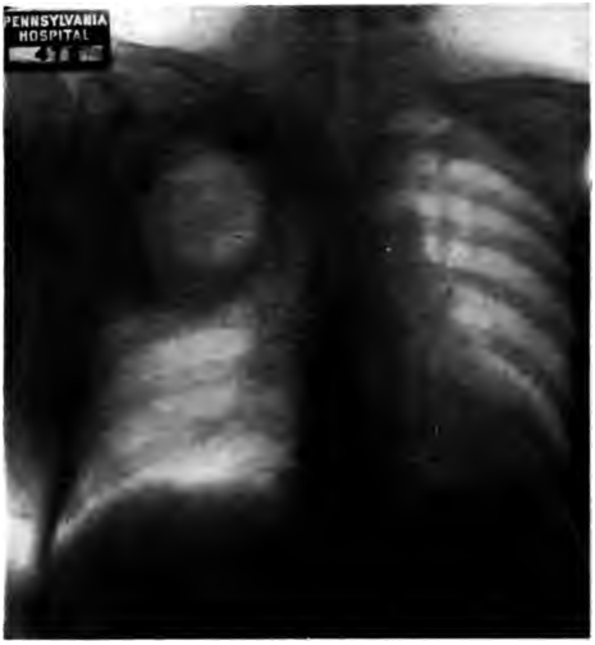

FIG. 314.—Same as Fig. 313, after spontaneous evacuation. (Anterior view.)

Symptoms.—An abscess is to be suspected, if in the presence of one of the known etiological factors, the patient develops respiratory symptoms or if already present, these symptoms become aggravated. The condition is also to be suspected in post-operative cases which develop respiratory symptoms and this is especially true if a tonsillectomy has been performed. In a typical case there is an intermittent type of fever (Fig. 315), chills, some sweating while the blood count shows a high leukocytosis. Cough and some expectoration are present and chest pain is not uncommon, especially if the pleura is involved. Blood-streaked sputum or a definite hemoptysis is an exceedingly common symptom.

ionally a fatal result is brought about by the occurrence of a large rrhage. Owing to the fact that the abscess is a secondary condition mptoms indicative of its presence may be masked or misinterpreted ason of the primary trouble.

ı the cases following pneumonia, cough, expectoration, fever and in the chest are already present. If the fever persists and is inter- nt with chills and sweats or fever develops after a decline, a pul- ry abscess should be thought of. It is to be borne in mind that a lung ss is more frequently encountered in the post-mortem room than bedside. In our series the symptoms were either not characteristic tirely lacking in two-thirds of the cases. The increasing experience roentgenologist is bringing to light many more of these abscesses was formerly the case. An X-ray examination should always be ed to in doubtful cases, more particularly to accurately locate the le.

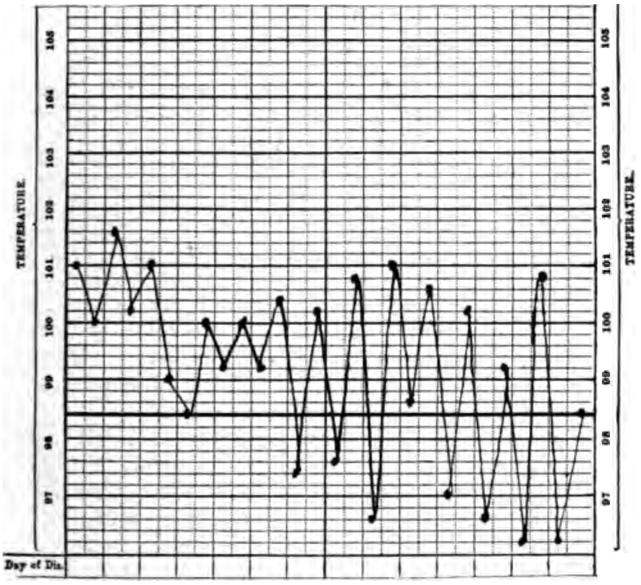

FIG. 315.—Pulmonary abscess.

he diagnosis is rarely made prior to or in the absence of the n expectoration of a large quantity of purulent sputum. This itutes the characteristic feature of pulmonary abscess and from this ymptom the diagnosis can be made with almost absolute certainty. sputum is often thick and ropy and pale yellow, bloody, reddish n or greenish in color. It may be odorless, slightly sweetish or very Following the initial outpouring of the pus, the process is repeated ervals. Each succeeding time the amount becomes less and less and y, in the favorable cases, ceases with the healing of the pulmonary .

ith the evacuation of the pus the constitutional symptoms cease, imes at once. If the drainage is inefficient and intermittent, the sputum becomes fetid and the constitutional symptoms, indicative of toxic absorption, more severe.

The sputum is negative for tubercle bacilli. The presence of elastic tissue points very strongly toward the presence of a pulmonary abscess. Its absence, however, does not rule out the presence of an abscess. By pouring the sputum on a piece of glass with a black background, pieces of lung tissue may be picked out. The elastic tissue appears as grayish-yellow spots which are selected for examination under the microscope. If there is no macroscopic evidence of broken-down lung tissue, the sputum should be stained with one of the elastic tissue stains, such as Weigert's. Fetor of the sputum or a foul odor of the breath is very common. The evidence of necrotic or gangrenous change in post-operative cases makes its appearance quite uniformly on the 13th or 14th day after the operation (Wessler, *loc. cit.*). Usually an aggravation of the symptoms is associated with an increase of the fetor and the expectoration of necrotic material and may be followed by temporary or permanent improvement.

The symptoms of a chronic abscess are not distinctive and the etiology is often obscure. Among the cases seen at the Phipps Institute suspected of having this condition there was a history of cough for months and the presence of purulent sputum which did not contain tubercle bacilli. The constitutional symptoms were slight and the general health fairly good. They were all referred to the dispensary as having tuberculosis. In two an X-ray examination indicated the probable existence of an abscess.

Physical Signs.—The physical findings in cases of abscess are not at all uniform. In those instances in which the abscess is secondary to pulmonary inflammation there are already present signs which may mask its presence. In three of the cases we have reported a pleural exudate obscured the signs.

The only distinctive features are the signs indicative of a cavity, *i.e.*, tympany on percussion, whispering pectoriloquy, amphoric or cavernous breathing and consonating râles. Any one or two of these signs may be wanting. Whether these signs can be elicited or not will depend on whether the abscess has emptied itself and formed a cavity and also on the location of the excavation. If empty and superficially placed the signs can be obtained easily. If the cavity is deeply placed in the center of the lower lobe, physical signs are wanting. As the majority of single abscesses are so situated it is not surprising that the signs of excavation are so frequently absent.

In 8 of the 30 cases in our series signs of a cavity were present. The area about the angle of the scapula is the most frequent site of the cavity signs. When located at the apex a sputum examination is the only means of differentiating the condition from tuberculosis. In the absence of cavity signs there are present an impaired or dull percussion note, increased tactile fremitus, distant bronchial or broncho-vesicular breathing and fine crepitating râles. The latter are due to the surrounding area of hyperemia and edema. Clubbing of the fingers may develop rapidly in some instances.

The signs of chronic abscess are those of some chronic inflammatory condition of the lung. In a case with a history of having had a cough for some time and in which there are signs of pulmonary damage a chronic cess is to be thought of. If the base of the lung is involved, tubercu-is is unlikely but in any event the sputum should be examined for ercle bacilli. An X-ray examination should always be resorted to erever possible.

Abscess of the lung, when definitely recognized, or even when only pected, belongs to that border-line group of cases in which the inter-t and surgeon should be associated from the beginning. From the ure of the trouble it is the internist who must recognize the trouble it is he who first sees such cases. There should be no delay in calling surgeon. When treated medically the *mortality* is about 50 per cent. ile among those treated surgically the mortality is about 25 per cent.

Diagnosis.—The two conditions with which a single abscess is most to be confused are *tuberculosis* and *bronchiectasis.* The acuteness the symptoms in abscess and the chronic nature of both tuberculosis d bronchiectasis are important points. The absence of tubercle bacilli, ecially in a case in which the symptoms have existed for some months, uld suggest the possibility of a chronic abscess.

MULTIPLE ABSCESSES OF THE LUNG

Etiology.—In 33 cases of multiple abscess encountered among the topsy records of the Pennsylvania Hospital for a period of fourteen rs the following etiological factors were noted:

Mastoid disease and thrombosis of the lateral sinus and internal jugular vein	1
Otitis media	2
Otitis media and thrombosis of the lateral sinus and internal jugular vein	2
Thrombosis of the portal vein and mesenteric veins and appendicitis.	2
Thrombosis of iliac vein	1
Broncho-pneumonia	6
Croupous pneumonia	3
Typhoid fever and broncho-pneumonia	2
Vegetative endocarditis infarct	6
Abscess of kidney	1
of lip	1
of pharynx	1
of seminal vesicles	1
of skin	1
Gunshot wound of chest	1
Not determined	2
Total	33

e exciting causes, as shown in the above table, are seen to differ from ose encountered in the case of a single lesion. In multiple-abscess rmation *supperative foci of the most diverse kinds and at distant parts of body play an important rôle.*

Another not infrequent cause of multiple-abscess formation is the as-ration of food or of septic material from an infected focus in another ea of the lungs.

Morbid Anatomy.—One or both lungs may be affected; the lower lobes e more frequently affected than the upper. The number of abscesses ll depend on the amount of aspirated material or the number of in-ted pulmonary emboli. The abscesses may be separated from each er by comparatively healthy tissue or may be confluent. The affected lung is usually congested and edematous and on section the abscesses are recognized as grayish-yellow, reddish areas. On cutting through one of the abscesses the center is apt to be broken down and occasionally a cavity is formed.

When the abscesses are due to metastatic or septic emboli the lung on section shows the infected foci in all stages from that of purulent infiltration to the formation of the abscess and excavation. Occasionally the lung presents a honeycombed appearance due to the formation of numerous small cavities.

Owing to the wide extent of the infection in the lungs and the serious constitutional disturbance, an individual suffering from multiple abscess formation rarely lives long enough to establish fibroid changes in the walls of the cavity. The condition is rarely chronic.

Symptoms.—A patient suffering from multiple pulmonary abscesses usually presents the clinical picture of pyemia. The fever is irregular and remittent or intermittent in type, there are chills and sweats, and every evidence of a severe infection.

A marked leukocytosis is usually present. The cough may be severe. The expectoration is sometimes profuse but rarely, if ever, occurs paroxysmally and in the large quantities characteristic of a single abscess. The sputum is purulent and not infrequently bloody. Elastic tissue may be detected in the sputum.

The presence of abscess formation is to be suspected in a patient who is suffering from some acute pyogenic infection, such as mastoiditis or acute otitis media, and who in addition develops pulmonary symptoms. The latter are not always present or, if so, may be so mild as not to attract much attention.

Physical Signs.—The physical findings are even more uncertain in multiple abscess than in the case of the single isolated lesion. In our series of 33 cases the following physical signs were noted:

Scattered râles	2
Scattered râles and friction rub	4
Scattered râles and areas of dulness	5
Areas of dulness	4
Dulness at one base alone	6
Signs of cavity	1
No record	11

The signs enumerated above may be elicited in a variety of pulmonary conditions. They are suggestive of abscess formation only when there is a known focus of suppuration; when there are symptoms indicative of a general septic infection; and when, in addition, the sputum is purulent or bloody in character and occurs in fairly large amounts.

Diagnosis.—The recognition of the presence of multiple abscesses is far from easy and their presence is unsuspected even oftener than in the case of a single abscess. In some instances their existence can be surmised in cases of general septicemia which develop respiratory symptoms or in cases of croupous or broncho-pneumonia in which a hectic type of temperature and chills and sweats succeed the initial trouble. When there are a number of abscesses involving both lungs or a considerable portion of one lung and there are no localizing signs the following conditions must be considered—tuberculosis or one of the mycotic infections. The examination of the sputum will rule out or establish the ɟnosis if due to one of these affections. If any of the abscesses break n, elastic tissue also may be present in the sputum.

PULMONARY GANGRENE

?or the first accurate account of pulmonary gangrene we are indebted ıaennec who described two forms: the circumscribed and the diffuse ;ircumscribed). His description of the morbid anatomy and the ptomatology is unrivaled to the present day.

ɜtiology.—Gangrene of the lung is one of the rarest of pulmonary :tions. Fowler in 1621 consecutive autopsies encountered but 7 nples of the disease (0.43 per cent.). Among 662 autopsies at the ɔps Institute 2 instances of pulmonary gangrene have been noted; of the diffuse form in a non-tuberculous subject and in the other gangrene complicated a tuberculous process (0.30 per cent.).

Ↄangrene of the lungs is never a primary condition but occurs second- r in a variety of conditions. When one comes to analyze the exciting ɛes of pulmonary gangrene, it is evident that there must be other factors ork besides necrosis of tissue and the action of saprophytic organisms, ;rwise the condition would be relatively common. As a rule gangrene ıcountered in individuals whose resistance has been lowered. This ' be due to a chronic debilitating illness such as diabetes; to the ex- ɜtion incident to certain of the insane states (dementia, melancholia) lastly to a lowering of vitality as the result of excesses.

Γhe following etiological factors have been noted:

l. It may be the terminal event in bronchiectasis and putrid bron- is. In both these conditions patches of septic broncho-pneumonia uently develop and these may become gangrenous.

:t has long been recognized that gangrene of the lungs not infrequently ırs among the insane. In these cases the exciting cause is an "aspira- " broncho-pneumonia. In the same manner a septic broncho- ımonia followed by gangrene, may occur secondarily to cancer of the ıth, tongue, pharynx or the esophagus. In rare instances gangrene ıe lungs is one of the sequels of a broncho-pneumonia complicating sles.

ls a sequel to croupous pneumonia gangrene is very infrequent and n it does occur it is usually in a debilitated or diabetic subject (Osler). , complication of pulmonary tuberculosis gangrene is rare. Only one ınce has been encountered at the Phipps Institute among 662 ıpsies. In two other cases the horrible fetor of the sputum lead to ıgnosis of gangrene but in neither case was it found at the autopsy. ne the fetor was due to an associated bronchiectasis; the other to ;mpyema which had ruptured into the lung and was discharging ugh a bronchus.

:. The pressure of a mediastinal tumor or aneurism may cause ɟrene. Such instances are rare, however. I recall one case in the :ce of the late Frederick A. Packard, at the Philadelphia General ɔital, in which the pressure of an aneurism of the descending aorta severed completely the left bronchus. The distal portions of the chus and the adjacent lung tissue had become gangrenous.

. Gangrene may develop as the result of an embolus lodging in a branch of the pulmonary artery. This sometimes occurs during convalescence from an attack of protracted fever, such as typhoid.

One of the main branches of the pulmonary artery may become thrombosed in which case the diffuse type of gangrene is produced. It may involve the greater part of one lobe.

4. Gangrene may be a sequel to some traumatic injury of the lungs.

From the above-etiological factors it will be seen that gangrene arises under much the same circumstances as abscess of the lung. In certain instances an abscess may later become gangrenous. It seems highly probable that the general resistance is a strong deciding factor. The more debilitated the patient the greater the likelihood of gangrene developing.

Morbid Anatomy.—*Circumscribed Form.*—The gangrenous focus may be single or there may be two or more. As in the case of abscess, the lower lobe is more frequently the site of the trouble than the upper. Walker[1] in an analysis of 40 cases found the right lung involved in 21; the left in 9; both sides in 2; and side not mentioned in 8. The gangrenous area is more apt to be located near the periphery of the lung than in the central portion.

Three stages are to be recognized: (1) The affected area is moist but firmer than the normal lung and usually greenish-black in color, though it may be of a dirty gray color. (2) The stage of liquefaction in which the affected area is converted into a fetid pulp. (3) The stage of excavation. The part of the lung immediately surrounding the gangrenous focus is deeply engorged and presents the appearance of hepatization; outside of this zone there is marked edema of the pulmonary tissue.

Laennec described the evolution of the process as follows: The affected area sometimes detaches itself from the surrounding parts forming a slough of a blackish, greenish, brownish or yellow color. This slough may remain isolated in the midst of the excavation formed by the destruction of the mortified part. It is more usual, however, for the eschar to be entirely softened without forming a separate slough, and to be transformed into a species of putrid pulp, of a dirty greenish-gray, sometimes bloody color, and of a horrible fetor. This softened mass soon makes its way through some neighboring bronchus and being, by degrees, evacuated forms a cavity.

With the separation of the eschar a cavity is formed with ragged and shreddy walls (Fig. 316) which are lined with a false membrane, of a grayish, or dirty yellow color and which secretes a dirty turbid pus or "black sanies." The lining membrane is soft and readily scraped off with a scalpel. If the cavity is small the false membrane may fill the space and be transformed into a cicatrix. The false membrane may be wanting. The wall is then composed of an infiltrated zone which secretes a foul pus.

In some instances blood-vessels entirely denuded and isolated, but perfectly intact, traverse the cavity. In other instances they are destroyed, the open ends being plugged with a thrombus. Occasionally a fatal hemorrhage results from the erosion of one of these vessels.

An associated bronchitis is nearly always present. Perforation of the visceral layer of the pleura may produce a pyo-pneumothorax. Occasionally a pleural effusion is noted. A hemorrhagic effusion occurred case at the Phipps Institute. In rare instances the gangrenous becomes cicatrized and the patient recovers.

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[1] *Boston Med. and Surg. Jour.*, July 9, 1914

ffuse Gangrene.—This form is very rare. It is characterized by sence of a limiting zone of inflammation the gangrenous area merg- to the surrounding lung tissue. Not uncommonly it involves a part of or even an entire lobe.

a case observed at the Phipps Institute the gangrene resulted from bosis of the branch of the pulmonary artery leading to the right

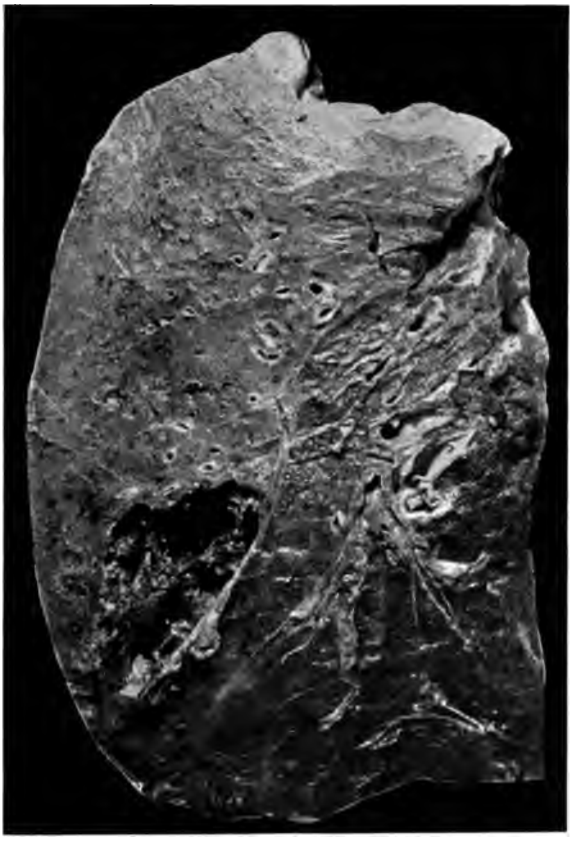

FIG. 316.—Gangrene of the lung. (*Philadelphia Hospital.*)

lobe. The affected lobe was dark red and very soft and friable, ecrosis and breaking down of the tissue had not occurred. There a number of areas in which the visceral pleura had disappeared. e place there was an infarct which was firmer and not so dark in as the surrounding tissue. The denudation of the visceral pleura esulted in a pneumothorax with a hemorrhagic effusion. The it had a hypertrophied and dilated heart.

e bacteriology of gangrene is varied. In addition to the pus- cing germs there are also numerous saprophytic organisms. It is ear whether gangrene can be produced by the saprophytes alone or whether an antecedent infection with the pus-producing organisms is also necessary. It is to be borne in mind that a number of cases of gangrene have been reported in which acid-fast bacilli have been found in the pus. Ophüls places these acid-fast organisms among the streptotricheæ.

Symptoms.—The symptoms of pulmonary gangrene may occur unheralded by any previous pulmonary trouble. As a rule, however, this is not the case and symptoms of a preëxisting pulmonary lesion are present. The premonitory symptoms vary greatly, depending, as they do, on such a variety of conditions.

The sputum constitutes the characteristic feature of pulmonary gangrene. It is of a horribly fetid odor which may permeate the entire house. If allowed to stand in a conical glass it separates into three layers: (*a*) a greenish-brown heavy sediment, (*b*) a middle layer of thin greenish or brownish fluid, and (*c*) a thick frothy top layer. The sputum invariably contains elastic tissue, and in some instances large fragments of lung tissue have been coughed up. While some observers claim that elastic tissue may be absent Osler states that he has never met with such an instance.

Cough is usually present and often paroxysmal in character. A frank hemoptysis is not unusual and may be the first sign. Pleuritic pain is very common. While the constitutional symptoms are usually severe, they are not distinctive of pulmonary gangrene. The temperature is irregular and of the hectic type; the pulse is rapid; and the prostration extreme. There is also a loss in weight although death usually occurs before the emaciation has progressed far. Death, generally ensues as the result of exhaustion; rarely a large hemorrhage is the terminal event.

If the gangrenous area remains closed and does not communicate with a bronchus the fetid sputum so characteristic of the disease will be absent. This so-called latent form is occasionally met with in the postmortem room in young children, in the insane and in diabetic subjects.

Physical Signs.—The physical signs are not distinctive. In the absence of the characteristic sputa they furnish no information as to the true nature of the trouble. In the first stage before breaking down of the pulmonary tissue has occurred, there is dulness or impairment of the percussion note over the affected area. Later, when excavation has occurred, the note may be tympanitic.

The breath sounds may be bronchial or very distant in character during the stage of engorgement. Fine moist râles are present. Later in the disease râles are heard over both lungs as the result of an associated bronchitis. Owing to the proximity of the affected area to the surface of the lung a friction rub is common. When the gangrenous eschar has been removed, signs of a cavity may be heard.

Diagnosis.—The characteristic feature of pulmonary gangrene is the indescribable fetor of the sputum. While some claim that the gar ous odor is distinctive it often cannot be differentiated from occurring in bronchiectasis, from that coming from an encysted emp which ruptures into the lung, or a pulmonary abscess.

In bronchiectasis there is an absence of elastic tissue. In the ca an encysted empyema or abscess the clinical distinction is not al easily made. The sputum may be horribly fetid in all three condit Elastic tissue occurs in both abscess and gangrene and may also be n in instances in which the pus ruptures into the lung. Even at the autopsy table the distinction between a pulmonary abscess and a gangrenous process is sometimes fraught with difficulty.

III

PULMONARY INFARCTION

(Pulmonary Embolism—Pulmonary Apoplexy)

Etiology.—In the recognition of this accident a knowledge of the etiological factors which predispose to it is of the utmost importance, as without such knowledge the phenomena, which attend the lodging of an embolus in a branch of the pulmonary artery, are apt to be incorrectly interpreted.

The source, from which pulmonary emboli come, are to be found either in the right side of the heart, or in the veins of the body. The reason for this is obvious. In order to reach the pulmonary circulation the embolus must, of necessity, pass directly from the right ventricle or from smaller to larger vessels in its course toward the heart, otherwise it would lodge at some distal point. On the other hand, an embolus finding lodgment in the other viscera such as the kidney, spleen, etc., travels in the opposite direction, and by way of the arterial system, its source being in the left side of the heart (simple or malignant endocarditis). A pulmonary embolism arises then under the following conditions:

1. *Valvular Heart Disease.*—Of the valvular lesions mitral disease is the most important, particularly mitral stenosis. Aortic lesions are rarely associated with pulmonary infarction. Pulmonary infarction due to cardiac lesions in the left heart is usually the result of stasis which leads to the formation of clots in the pulmonary artery.

When the embolus originates in the right heart the process is as follows: Fibrin is deposited on the auricular or ventricular walls and this, in addition to slowing of the circulation, leads to coagula forming in the interstices of the muscular bundles. Eventually fragments are broken off and escape into the pulmonary circulation.

2. *Phlebitis.*—The occurrence of a thrombus in some portion of the venous system is the most prolific source of hemorrhagic infarction of the lung. Venous thrombi occur not infrequently in the acute infections, especially typhoid fever. Of the veins involved those of the lower extremities, especially the femoral, are perhaps most frequently affected. A venous embolus may consist of a large fragment from a thrombus in one of the large veins, such as the iliac or femoral. Owing to its size it produces an infarct of large size and if death does not ensue immediately, the physical signs resemble those obtained in cases of croupous pneumonia. Large venous emboli are not common. On the other hand, small venous emboli are not of infrequent occurrence. They result from the separation of small fragments during the formation of the thrombus and may manifest themselves by pulmonary symptoms days before the presence of the thrombus is known to exist. Probably the best known form of venous thrombosis is that occurring in the puerperal period, and commonly referred to as phlegmasia alba dolens or milk leg. In addition it must be borne in mind that pulmonary emboli not infrequently come from a thrombus formation in one of the uterine veins following confinement. This usually occurs in the first three weeks of the puerperal period; beyond the fifth week its occurrence is exceptional.

Of recent years it has come to be a well-recognized fact that a pulmonary embolism may follow various operations, particularly those on the abdominal or pelvic organs. It is more than probable that not a few cases of pneumonia, following major operations, are in reality instances of pulmonary infarction. Venous thrombosis may occur in chlorotic girls, in those who are gouty, as a complication of varicose veins and in those who are apparently healthy. Conner[1] has reported six cases of pulmonary infarction due to venous thrombosis, occurring in apparently healthy individuals.

3. *Infected Emboli.*—The most serious form of pulmonary embolism is that in which the thrombus harbors some microörganism. In these cases there is added to the danger of the infarct the certainty that it will develop into an abscess. Multiple abscesses of the lung, having their origin from an infarct, are almost constantly associated with general pyemia or right-sided malignant endocarditis.

4. *Fat Emboli.*—Although not of venous origin, a pulmonary infarct may be produced by a fat embolus following fracture of one of the bones. As a rule the fat globules lodge in the finer capillaries, and rarely obstruct a branch of the pulmonary artery, sufficiently large to produce symptoms. Bissel has reported, from the Mayo Clinic, the finding of fat emboli in the lungs following operations. The emboli may be very small. They are associated with broncho-pneumonic areas but it is uncertain whether they cause the pneumonia or not. Cyanosis is the most constant symptom of the condition. A number of reports have pointed out the frequency of pulmonary fat embolism following wounds involving fatty tissue, the liver and more particularly, wounds producing compound fractures. In many instances the symptoms resemble those produced by "shock." Associated with pulmonary fat embolism, disseminated fat is also found in other tissues of the body.

5. *Foreign Material.*—In all forms of hypodermic medication it is usual to emphasize the importance of being sure the needle is not in a vein, otherwise the injected material might be carried directly into the pulmonary artery, and be followed with serious results. An instance of this has come to my notice. Almost immediately following an injection of gray oil into the buttock, the patient presented all the classical phenomena of pulmonary embolism, and although he recovered, his life was despaired of for some hours.

6. *Air Emboli.*—Lastly we have to consider the occurrence of air emboli. For years the possibility of this accident has been repeatedly emphasized in carrying out procedures which involve injecting any substance into the veins. As a matter of fact, however, the occurrence of such an accident has never been proven, and there are those who disbelieve entirely in there being any likelihood of such an accident.

Morbid Anatomy.—When an embolus is detached from the pare thrombus it is at once swept along the venous blood current. As i passes through vessels which are constantly increasing in size, no obstru tion is offered in its journey toward the right side of the heart. O leaving the right heart, the embolus enters the pulmonary artery, here the conditions are reversed, as the branches of the pulmonary art are terminal vessels. Depending on the size of the embolus a vessel r or later reached, which is too small to admit of its further passage. lock usually occurs at a point where the artery bifurcates. With lugging of the vessel the pressure in the area situated peripherally e blocked point almost disappears. This results in a backward f blood, which stagnates in the affected area, and in addition the s elements of the blood leak through the walls of the vessel, thus ting the tissues in the area supplied by the plugged vessel. The o affected constitutes the so-called ~~hemorrhagic~~ infarct. If there

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[1] *Arch. Int. Med.*, March, 1914.

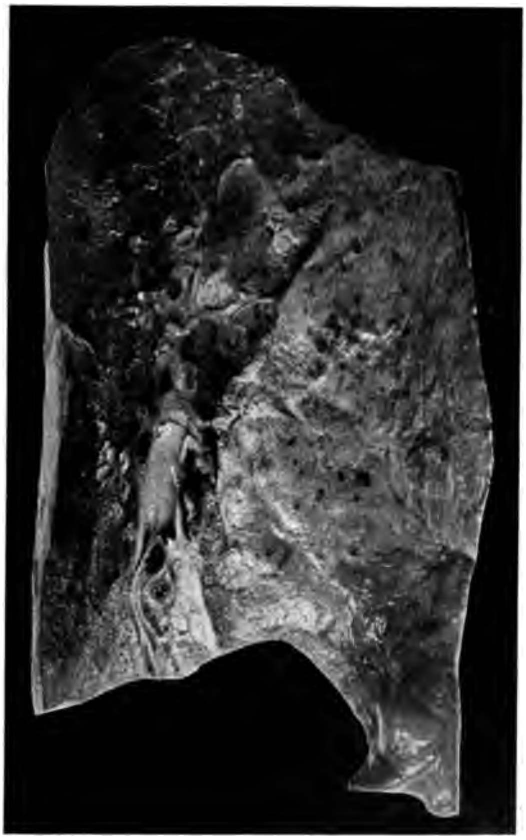

FIG. 317.—Multiple infarction of the lung.

en a preëxisting stasis in the pulmonary vessels, such as is met with onic cardiac disease, the hemorrhagic character of the infarction es more marked.

lmonary infarcts are more commonly met with in the lower than per lobes of the lung. They are conical or wedge-shaped, the base formed by the visceral pleura. The affected area generally pro- lightly above the surface, the visceral pleura being covered with a us deposit, which may be limited to the area involved, or extend distance about it. The extent of the infarct depends on the size of the vessel which has been occluded; it may correspond to the area of one or two lobules (Fig. 317), or may involve nearly an entire lobe.

On section, the wedge-shaped infarct is readily distinguished by the black-red color, and the fragile condition of the blood-soaked tissue. Pulmonary infarcts may be entirely absorbed, but as a rule resolution is not complete and a scar results. If the infarct is of septic origin as in cases of right-sided malignant endocarditis or general pyemia, abscess formation results.

Symptoms.—The severity of the symptoms, following the lodgment of an embolus in the lung, depends on the size of the vessel blocked.

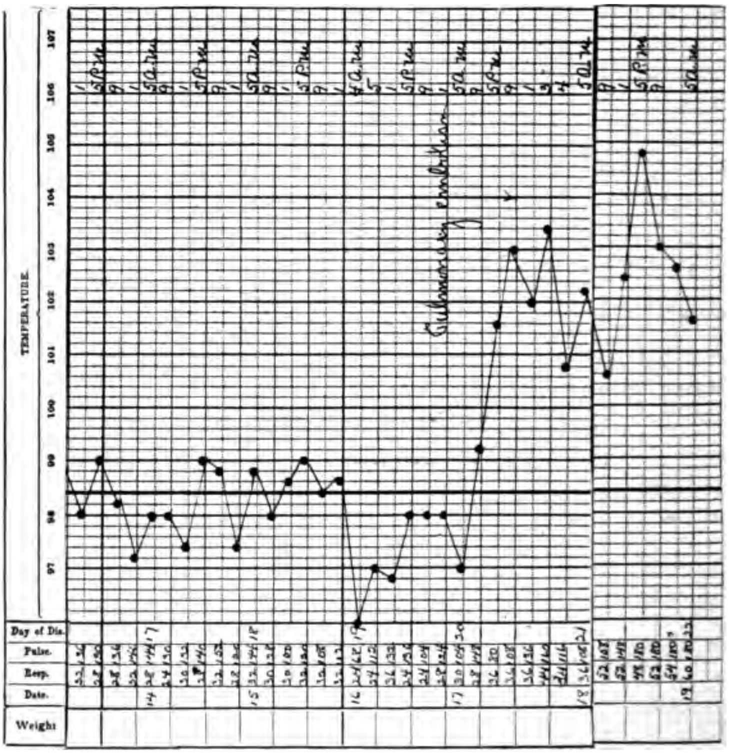

FIG. 318.—Pulmonary embolism in a patient convalescing from typhoid fever. (Interval of one day before the rise of temperature)

If the embolus is a large one, the patient may be so severely shocked that death occurs immediately or within an hour or two. On the other hand, the embolus may be so small that it produces no symptoms at all. The mechanism by which death is produced by an embolus which blocks only a small part of the pulmonary blood circulation is unknown. Mann[1] in an experimental study was unable to produce death until the pulmonary circulation was practically occluded. When a moderately large branch of the pulmonary artery is suddenly occluded, the patient is seized wih sudden dyspnea, cyanosis and intense pain in the side. This is followed a few hours later by the expectoration of liquid or dark red clotted blood, which is not frothy, as in the hemoptysis of phthisis. An initial chill is rarely observed. Strümpel states that fever is apt to be delayed a day or so following the formation of the infarct (Fig. 318), and that this is sometimes of service in distinguishing the condition from lobar pneumonia.

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[1] *Jour. Exper. Medicine*, Sept., 1917.

In those patients in whom the symptoms are overwhelmingly severe, the source of the embolism is apt to be in the veins, while the less severe instances are met with in cases of chronic cardiac disease.

Physical Signs.—The recognition of a pulmonary infarction depends, primarily, on a knowledge of the etiological factor, and secondarily, on the symptoms. Physical signs at best will indicate only the size of the involved area. If, however, the infarction is small or centrally located, chest signs will be entirely wanting.

Inspection.—So far as the chest is concerned, inspection will be negative unless a large portion of a lobe is involved. If that occurs, expansion of the affected side will be diminished.

Palpation.—Palpation will reveal deficiencies in expansion, if any such exist. Vocal fremitus will be increased providing the infarct is sufficiently large, otherwise no change is apt to be detected.

Percussion.—Impairment or absence of pulmonary resonance will also depend on the size of the infarct, and whether it is peripherally located. The area of dulness does not extend as in pneumonia and if small and circumscribed usually disappears in two or three days. If the infarct is large, the percussion note may be flat and suggest the presence of an effusion. Dulness may change later to tympany if the infarct breaks down and an abscess results.

Auscultation.—In cardiac cases râles, both large and moderate-sized, are apt to be present throughout both lungs as the result of stasis. Localizing signs, such as fine crackling râles, or a friction rub, are not likely to be present for some time after the formation of the infarct and may be the only signs present. The breath sounds, however, are apt to be absent or very distant from the beginning. Later, if the effused blood is absorbed, the breath sounds return, and the râles become coarser over the involved area. In septic cases the infarct may change into an abscess and empty into a bronchus, thus forming a cavity. Under these circumstances the signs over the affected area differ in no particular from cavity formation due to other causes.

Diagnosis.—Cases in which there is an evident cardiac lesion or the presence of a phlebitis offer little difficulty in diagnosis. In those cases in which the phlebitis is latent or is located in veins inaccessible to sight or touch, the source of the respiratory symptoms may be difficult to interpret. The sudden appearance of symptoms and signs due to a pulmonary infarction may be the first intimation we have of a venous thrombus.

The condition is to be distinguished from *pulmonary tuberculosis* largely by the absence of tubercle bacilli and the location of the signs at the bases rather than the apices of the lungs. In addition more than one area may be involved in cases of infarction. If the infarct is deeply seated and does not give rise to physical signs, the differentiation may be very difficult.

Owing to the sudden onset of the symptoms with dyspnea, pain in the side and bloody sputum, a pulmonary infarct may be mistaken for *lobar pneumonia.* In the case of an infarct an initial chill is rare; fever may not appear immediately, and when present does not resemble that seen in pneumonia. The sputum consists of liquid blood or dark red clots and is not viscid.

In *malignant disease* of the lungs the sputum sometimes consists of dark clots resembling currant jelly. The presence of a primary tumor elsewhere should make the cause of the blood spitting apparent. In any event the course of the disease will not be that of infarction which tends to recovery.

PULMONARY CONGESTION

The term pulmonary congestion is loosely applied to a hyperemic condition which accompanies or follows other morbid conditions affecting the lungs or heart. Two forms are described—the acute and the passive.

ACUTE CONGESTION

Etiology.—Hyperemia of the pulmonary tissue may occur in a variety of conditions. It constitutes part of the pathological picture in all acute inflammatory affections of the lungs, especially the first stage of lobar pneumonia. It is also noted in varying degrees in the pulmonary tissue surrounding recent tuberculous deposits.

Severe overexertion is sometimes attended by an intensely hyperemic condition of the lungs which, in a few instances, has been believed to be the cause of sudden death following athletic competition.

Areas of acute pulmonary congestion are sometimes noted in association with the acute fevers, notably, typhoid, influenza and malaria.

A very frequent cause of acute pulmonary congestion is the inhalation of irritant gases. If the exposure is slight, recovery ensues but if the individual is subjected to a large dose of the gas, death very frequently ensues. Oliver has noted intense congestion of the lungs as the result of the inhalation of carbon monoxide and sulphuretted hydrogen gases and from the fumes of nickel and ferri carbonyl and nitrous and nitric acid. He has also called attention to the fact that a workman who inhales chemical fumes may not exhibit any symptoms immediately, and yet within 24 to 48 hours he dies of acute congestion of the lungs. During the present European war chlorin and other gases have been used extensively as offensive weapons. The effects produced by the inhalation of irritant gases are described on page 521.

Exposure to extreme cold or the inhalation of very hot air may give rise to congestion of the lungs.

Woillez has described a primary form of pulmonary congestion which occurs without known cause. While the existence of a primary form is strongly supported by French writers, most authorities do not recognize it. The onset, the symptoms and the physical signs are similar to those occurring in lobar pneumonia and it is more than likely that it represents a mild or abortive type of the latter disease. It has a duration of but four or five days and almost invariably terminates in recovery.

Morbid Anatomy.—The amount of blood in the pulmonary vessels depends on the physiological activity of the lungs. Hyperemia may occur under normal conditions. If, however, a hyperemic condition arises as the result of abnormal conditions, it constitutes a congestion.

Congestion of the lungs is the initial stage of an inflammatory process and is usually followed by an exudation. It is not always easy, therefore, to draw a line between what constitutes simple congestion and that which may be considered edema. Acute congestion may be bilateral or it may be limited to one lung or a portion of one lung. The involved portion contains an excess of blood due to the distended capillaries. On section the lung resembles what may be seen after death from pneumonia, when the disease has involved parts which are crepitant and not yet consolidated. The affected areas are bright red, crepitant, feel dry to the touch and there is very slight if any perceptible increase of specific gravity.

Symptoms.—Pulmonary congestion usually gives rise to some dyspnea, cough and the expectoration of frothy, viscid or mucopurulent material. The sputum may be streaked with blood or show the presence of red blood cells microscopically. Unless the congestion is marked and associated with a slight amount of exudate, the sputum may be very scanty or entirely absent. Congestion due to the inhalation of irritant gases produces the most marked symptoms and in addition to the dyspnea and cough, there are marked irritation of the upper respiratory tract and a feeling of soreness and contraction in the chest.

PASSIVE CONGESTION

Etiology.—The underlying cause of passive congestion is to be found, almost invariably, in some cardiac affection. Probably the most common form of congestion of the lungs is that known as *hypostatic*. It is very frequently present at the bases of the lungs in individuals who have been confined to bed for a long time because of some protracted febrile disease, such as typhoid or other long debilitating illness. It may occur also in association with cerebral lesions, notably those which produce paralysis or coma. It is occasionally encountered in cases of morphine poisoning. Two factors enter into the causation of hypostatic congestion, namely, a weakened heart muscle and gravity. That a weakened heart muscle is the chief cause is evident from the fact that a healthy individual may lie in bed for an indefinite period without developing circulatory disturbance in the lungs.

Another form of passive congestion is that due to prolonged stasis of the blood in the pulmonary circulation. A weak heart muscle, whether it be due to myocarditis or dilatation secondary to valvular disease, leads to interference with the return of the blood from the lungs to the heart. Although any form of cardiac disease may cause passive congestion, the most extreme grades of the condition are seen in cases of mitral stenosis.

Passive congestion is sometimes encountered as the result of obstruction to the flow of blood through the pulmonary capillaries. Under those circumstances it is often encountered in cases of chronic emphysema and fibroid affections of the lungs. Thrombosis of the pulmonary veins or compression of the pulmonary veins by an aneurism or tumor may also produce the condition.

Morbid Anatomy.—Hypostatic congestion occurs, as a rule, in the posterior portions of the bases of the lungs or in the dependent portion of one lung if the patient has lain on that side. The affected portion is dark red or purplish red in color, heavier than normal and on section drips blood or bloody serum. Owing to the fact that there are commonly associated with the congestion varying degrees of edema the lung pits on pressure.

In those cases in which the engorgement of the blood-vessels is marked and there is, in addition, some leakage of the blood into the vesicles, the condition is sometimes referred to as hypostatic pneumonia or splenization of the lung.

A lung which has been the seat of a passive congestion due to stasis, of long standing, is somewhat enlarged, is less elastic and cuts with greater resistance than normal. It is of a dark red or purplish-red color and on section is found to be full of blood of a venous color. The engorged condition of the lung is due to stasis of the blood current in the pulmonary circulation. In mitral stenosis, for instance, the pulmonary vessels are, on the one hand, distended with blood which is dammed back owing to the obstruction at the mitral orifice, and on the other hand, by blood which is forcibly driven into them by the hypertrophied right ventricle. This gradually leads to a proliferation of the fibrous tissue in the septa of the lung. If the condition persists, the continued tension within the capillaries leads to the escape of red blood cells by diapedesis. The blood cells eventually break down and liberate the blood pigment. This produces a condition known as *brown induration* or "heart lung." In such a lung there is a marked increase in the fibrous tissue and the organ is of a dark, rusty brown color. On section a brownish colored fluid can be squeezed out. Microscopically this fluid is seen to contain large mononuclear cells containing brownish pigment. These cells, sometimes referred to as "heart failure" cells, may be found in the sputum also. In common with other forms of pulmonary fibrosis there may be some dilatation of the bronchi.

Symptoms.—There are no symptoms which are characteristic of hypostatic congestion. It may give rise to some dyspnea, slight cyanosis and cough. As a rule, its presence is detected in the routine examination of patients suffering from typhoid fever or other adynamic disease.

Passive congestion due to heart disease does not produce symptoms so long as cardiac compensation is maintained. The sputum, however, may show the presence of the characteristic pigmented "heart cells." The first evidence of failing compensation may be the appearance of dyspnea and cough, especially after the slightest exertion. Sputum may be absent or moderate in amount and when present may be blood-streaked. Small hemoptyses may occur also. Sudden weakening of the left ventricle may be followed very quickly by marked dyspnea, cyanosis, cough and the expectoration of blood-streaked sputum. The condition is often referred to as "cardiac asthma."

Physical Signs.—In the presence of hypostatic congestion examination of the lower and posterior portion of the lungs will usually show some impairment of the percussion note, more or less suppression of the respiratory murmur and the presence of medium-sized and crepitant râles.

Passive congestion due to heart disease does not give rise to physical signs unless cardiac compensation fails. In the event of this happening mixed râles are heard over both lungs especially at the bases.

PULMONARY EDEMA

This condition is, as a rule, a sequel of congestion and differs from the latter only in the exudation which is present. Like congestion, edema occurs in an acute and a chronic form.

The cause of pulmonary edema is not altogether clear. The generally accepted belief is that the exudate arises as the result of a mechanical defect which is cardiac in origin. This conception is based on the classic experiments of Welch who believed that the edema resulted from stasis due to increased capillary tension. The tension is brought about by an excessive amount of blood in the pulmonary capillaries, due, in part, to a weakened left ventricle which dams the blood back, and, in part, to an excess of blood forced out of the right ventricle. As a result of this overfilling of the pulmonary vessels, serum escapes from the distended capillaries. This explanation is entirely applicable to the so-called terminal edema which, for the most part, is associated with cardiac or cardiorenal disease.

Sahli attributes the edema to changes in the capillary vessels. This hypothesis is supported by Krehl who points out that the edema is unevenly distributed and often definitely associated with inflammatory processes. It is well known that it is often difficult and may be impossible, to distinguish between areas of edema and pneumonic patches. Weakening of the capillary walls may be produced either by a chronic disease such as nephritis or by an acute inflammatory process.

Edema may occur also after general anesthesia or in the course of one of the acute infectious diseases. In these cases it is believed, by some, that the edema is toxic in origin especially in those cases in which there is no associated cardiac lesion to account for the condition. In support of this view is the well-known fact that experimentally the injection of adrenalin will produce, in animals, a condition comparable to the acute edema seen in human beings. Kraus[1] from experimental observations, states that disturbance of the nervous mechanism governing the pulmonary circulation may produce edema.

"On the whole, the most acceptable view as to the etiology of pulmonary edema is that it is due to increased capillary tension accompanied, aided, and probably in many instances preceded, by degenerative changes, toxic in character, of the capillary endothelium" (Hare).

CHRONIC PULMONARY EDEMA

Etiology.—Localized pulmonary edema is met with under a variety of conditions. Occurring in the pulmonary tissue surrounding circumscribed lesions, it forms part of the pathological picture in broncho-pneumonia, abscess, new growths, infarcts and tuberculosis. General edema of the lungs often occurs in chronic conditions, such as cardiac disease, nephritis, anemia, and cerebral disease. As a terminal event it is not uncommon. Cohnheim has said of this type that patients do not die because of the edema, but the edema develops because the patient is dying. In the great majority of cases the terminal edema is due to cardiac failure. Coplin found that cardiac disease was the cause of the terminal edema in 350 of 405 cases in which the condition was found at autopsy.

Morbid Anatomy.—General pulmonary edema is usually bilateral and most marked at the bases of the lungs. If, however, the patient lies constantly on one side the edema may be limited to the dependent lung. The condition is sometimes referred to as hypostatic pneumonia. The affected portion is heavier than normal, pits on pressure and except in extreme cases, is slightly crepitant. On section the involved portion is pale unless there has been marked congestion, when the cut surface is reddish in color. The cut surface may present a glistening gelatinous appearance if the edema is extensive.

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[1] *Berl. Klin. Woch.*, i, 1035, 1913.

The characteristic feature is the outpouring of a serous exudate into the alveoli and interstitial tissues. When the lung is incised there pours out a clear, frothy serum. If, however, congestion has been marked the serum is of a pinkish or reddish color.

Symptoms and Physical Signs.—The symptoms are essentially the same as those encountered in chronic congestion. In patients suffering from a prolonged illness, examination of the bases of the lungs posteriorly will often show impairment or even absolute dulness of the percussion note; suppression of or absent breath sounds; diminished voice sounds: and fine râles. If the edema is extensive, the air cells and bronchi may be filled with the exudate. In such cases râles are absent, the percussion note is dull and the breath and voice sounds very distant. The condition may be mistaken for a pleural effusion. The introduction of a needle is often needed to determine the presence or absence of fluid.

ACUTE PULMONARY EDEMA

This condition is rarely if ever primary in origin. Owing to its recurrent character Stengel[1] has suggested the term paroxysmal pulmonary edema. It is most commonly encountered in individuals suffering from some form of cardiovascular disease, particularly angina pectoris and myocarditis, or chronic Bright's disease associated with marked arterial hypertension. Occasionally in cases of pneumonia or pulmonary tuberculosis a fatal termination may occur suddenly with the development of an acute edema involving a part or all of the lung not involved in the original trouble. Less frequently it is met with in association with some one of the acute infections, pregnancy, epilepsy, anesthesia, or as one of the manifestations of angio-neurotic edema. A few cases have been recorded in which the condition followed the removal of a pleural or abdominal effusion. Acute pulmonary edema may end fatally in a few hours; on the other hand, there may be a succession of attacks occurring at more or less frequent intervals for years. Two cases which have come to my notice have had repeated attacks, one because of the associated external phenomena was undoubtedly *angio-neurotic* in origin; the other while apparently idiopathic, had in all probability, a similar basis.

Morbid Anatomy.—In those cases in which a fatal issue has occurred, the lungs are found to be heavy, water-logged and pit on pressure. When cut, an excessive amount of clear or pinkish-colored serum exudes.

Symptoms.—The attack comes on very suddenly with little or no premonitory indication and rapidly assumes such an alarming character that the patient and those about him feel that a fatal termination can be a matter of but a few minutes. At the moment of the attack the patient grows suddenly deathly pale and has an expression of terror. The hands, feet and whole body grow cold and become bathed in a dripping sweat. There is a feeling of oppression in the chest, extreme dyspnea or even orthopnea, and a short, incessant cough. Large quantities of clear or faintly blood-tinged, frothy serum are expectorated. In extreme cases e frothy serum may be expelled in a sudden gush through both the outh and nose. In individuals suffering from chronic cardiac or renal isease the pulse is very weak, and it is in these cases that a fatal issue is be feared. The sudden onset, labored breathing, the pallor, the cyanos, and above all, the character of the expectorated material renders the iagnosis easy almost at a glance.

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[1] *Amer. Jour. Med. Scs.*, Jan., 1911.

Physical Signs.—There are no signs especially characteristic of this ondition.

Inspection.—Aside from the labored character of the breathing, there re no noteworthy signs referable to the chest.

Palpation may reveal a rhonchal fremitus, although this is not apt o be the case, as the râles are of the fine sibilant and subcrepitant type.

Percussion is negative, except in the fatal cases where the outpouring f the serum is so great as to produce impairment of the pulmonary esonance.

Auscultation.—The breath sounds are for the most part obscured by he numerous fine sibilant and fine bubbling râles. The heart sounds are bscured for the same reason, and also because of weakness on the part of the heart itself.

EFFECTS OF POISONOUS GASES ON THE RESPIRATORY TRACT

The rapidity with which poisonous gases act when inhaled can be readily understood when there is taken into consideration the fact that they are diffused in the same manner as the inspired air and hence come into contact with the enormous absorptive area of the lungs which includes myriads of capillaries. So far as the respiratory tract is concerned our chief interest is with the irrespirable gases. Exposure to these gases has always constituted one of the industrial hazards and the literature contains a number of references to the subject. The extensive employment of irrespirable gases in the recent war was one of the most serious problems the medical officers had to deal with and while it is earnestly to be hoped there will never be a recurrence of this wicked method of conducting war, the effects of these gases should be understood because of the possible after effects they may cause even in men who have apparently recovered from the initial exposure.

I am indebted to my colleague, George W. Norris, for the following account of the pathological and clinical effects of the various types of gas employed in the recent war.

THE DIFFERENT TYPES OF GASES USED IN WARFARE

1. *Lacrimators* (acetone, xylene or benzene bromid).—These gases ct immediately, causing a copious ocular irritation, intense lacrimation nd thus a temporary, indirect functional blindness. They are comonly employed in gas chambers for purposes of instruction, and to test ut the "fit" of a mask.

2. *Sternutators* (diphenylchlorarsin).—The symptoms produced by is type of gas are immediate sneezing, coughing, headache, salivation, miting and substernal pain. They are employed preliminary to early in a gas attack so as to render gas mask protection difficult impossible. A man who is seized with the above mentioned sympotms

generally finds it impossible to get or keep his mask on, and hence fall a victim to the lethal gas that accompanies or follows the initial dose.

3. *Lung Irritants* (suffocating gases—chlorin, phosgen, diphosgen oxychlorcarbon).—These gases produce death by irritation of the pulmonary alveoli, resulting in edema of the lungs.

4. *Vesicants* (dichlorethylsulphid, dichlormethylether).—The brunt of the attack is borne by the skin, eyes and upper respiratory tract. Blistering and subsequent sloughing of the tissues affected results in cutaneous burns and intense bronchial, tracheal and laryngeal inflammation, and sloughing. Death results from broncho-pneumonia.

5. *Gases Destroying Erythrocytic Function.*—(*a*) Carbon monoxid—causing death by asphyxia resulting from methemoglobinemia.

(*b*) Arsin—causing hemolysis, jaundice, hemoglobinemia, nephritis and gastro-intestinal disturbances.

Diphosgen (trimethylchloroforminate, "surpalite").—This gas, which was used instead of phosgen ("palite"), is the most intense lung irritant known. In a dilution of 1 :50 (rare) one inhalation kills by laryngeal spasm. It diffuses quickly, and in a dilution of 1 : 1,000,000 is toxic if breathed for a considerable time. When brought into contact with water it forms hydrochloric acid. It produces but little local or early irritation, and during periods of excitement or when mixed with smoke, may be breathed for some time without discomfort. It does, however, cause slight, early, ocular smarting, nausea, retching and vomiting (pharyngeal irritation). Its chief and important action, however, is on the pulmonary alveoli, and its effect is *pulmonary edema.*

Morbid Anatomy.—The trachea and bronchi are filled with slightly blood-tinged froth. The lungs outwardly appear mottled as a result of alternating areas of congestion and emphysema. On section, congestion, capillary hemorrhages and extreme widespread edema are found. Small, slightly bloody pleural effusions are quite common. The right heart is dilated and often contains thrombi. The venous system is engorged, and thromboses are sometimes encountered. The kidneys are congested, and the glomeruli may contain clots.

Four-fifths of all the deaths occur within the first twenty-four hours. If the patient can be tided over this interval he usually recovers. The pulmonary edema clears up rapidly after the second or third day. Cardiac dilatation may persist and be troublesome for several weeks.

Symptoms.—These are slight cough, nausea, substernal pain, soreness or sense of constriction, headache, epigastric pain, dyspnea, fever from 100° to 106° F., syncope and weakness.

Slightly gassed cases are difficult to diagnosticate. The symptoms may be slight and physical signs absent. It is safe to assume that a man who shows no physical signs at the end of from twenty-four to thirty-six hours either has not been gassed or, if so, only to a negligible degree.

The severe cases fall into one of two categories: (*a*) *cyanotic cases:* increasing restlessness, dyspnea, weakness and cough with slight frothy, sometimes blood-tinged, expectoration; dyspnea. cyanosis, venous distention and a full, strong pulse of about 100; or (*b*) *gray cases:* slight cough, no expectoration, ashen pallor, marked tachypnea, shallow respiration, a leaky skin, very low blood pressure, a rapid thready pulse (140) and collapse. Needless to say, the prognosis in the latter case is far worse than in the former.

Physical Signs.—In the early stages there may be no auscultatory signs—simply an increase of the respiratory rate and an increased fulness of the pulse. Later the signs of beginning pulmonary edema manifest themselves—crackling râles, harsh breath sounds, not infrequently together with pleural frictions. It is noteworthy and striking that such physical findings are first noted, most marked, and, during convalescence, most persistent, over the upper lobes anteriorly, and not, as is the case in disease, at the bases. Local hyperresonance may be found, resulting from emphysematous areas. As the edema increases, resonance becomes impaired and breath sounds are lost, being totally replaced by crackling râles or completely silent areas. The heart dulness increases to the right, and as dilatation increases, the pulmonic second sound becomes enfeebled. Death results from deficient oxygenation due to cardiac dilatation, following pulmonary engorgement and edema. It is mechanical, therefore, there being no direct cardiotoxic effect. Occasionally symptoms and physical signs may be precipitated by physical exertion or a full meal, several hours after the exposure to gas, the patient collapsing quite suddenly.

Dichlorethylsulphid (yperite, "mustard gas").—This gas has a faint odor which has been compared to French mustard, vinegar, etc. Characteristically, it produces no immediate or early effects. One may remain or sleep in a dugout charged with it for several hours before becoming conscious of its presence, and then it is often too late.

Morbid Anatomy.—One finds purulent inflammation, destruction and sloughing of the respiratory mucosæ. The lungs show alternating areas of congestion, emphysema and broncho-pneumonia. The small bronchi and bronchioles are filled with pus. Sometimes coalescing areas of broncho-pneumonia may produce extensive lobar involvement. Small areas of pulmonary edema may exist. The stomach, chiefly near the cardia, as well as the duodenum, generally contains submucous hemorrhages, conditions apparently resulting from the swallowing of saliva and responsible for the epigastric pain and gastro-intestinal symptoms. The kidneys are usually congested. Death results from broncho-preumonia and asphyxia due to obstruction of the smaller bronchi and bronchioles by mucosal sloughs and pus.

Symptoms.—Occasionally nausea, retching, vomiting, and smarting of the eyes occur as valuable, even life-saving, symptoms at the end of from twenty to sixty minutes. As a rule, however, symptoms appear from two or five hours after the beginning of exposure.

The face and neck become red and the eyelids swollen. There is marked photophobia and blepharospasm, lacrimation, ocular and substernal and epigastric pain, pharyngitis, bronchitis, and an increased pulse and respiratory rate. At the end of twenty-four hours, severely gassed patients show in addition to an exaggeration of the symptoms just mentioned, cutaneous blistering of the face, neck, scrotum, inner surface of thighs, penis, buttocks, etc. Burns in the latter region are especially common after sitting on gas-impregnated earth. Widespread and severe purulent inflammation of all the upper mucosæ and the bronchi are present. The expectoration consists of bloody pus and is sometimes accompanied by sloughs of the entire tracheal and bronchial mucous membrane.

At the end of forty-eight hours, broncho-pneumonia, often widespread and coalescing, has occurred, as a result of tissue destruction and leukopenia. The patient is delirious, coughs, is cyanotic, exhausted, tortured by pain and suffocation, and finally lapses into unconsciousness. It is to be noted that in contrast to the phosgen cases, dyspnea, cyanosis, fever, tachycardia and prostration occur late. Practically none of the patients die within thirty-six hours, and death may occur weeks later. Slight transient albuminuria is quite common.

Convalescence.—Photophobia of a functional type and unexplained origin persists for weeks and should be ignored as soon as local inflammatory changes have subsided. Bronchitis may also last for weeks, together with substernal and epigastric pain. None of these symptoms should prevent a man's being sent to the convalescent depot if his general physical condition warrants it. Some of the cases develop E. S. (effort syndrome), and require special treatment in "heart classes." Severely gassed patients—those that have developed a demonstrable bronchopneumonia—usually die. The slightly gassed patients usually recover. There is doubtless a middle class in which bronchiectasis develops in time as a result of cicatricial changes; but so far as is known, true tuberculosis as a sequel does not occur. It is, of course, easy to conceive that latent lesions may be activated by gassing.

Ethyldichlorarsin.—This gas and others containing arsenic were increasingly used toward the close of the war. Shells containing 50 per cent. dichlormethylether and 50 per cent. ethyldichlorarsin were also employed. Aside from early sneezing, this gas produces a condition practically identical with yperite except for the fact that its action is much more rapid. It is said that the arsenical gases, in addition to the other symptoms, produce somnolence, nephritis (edema of the hands and face), abdominal cramps, diarrhea and hemolytic jaundice.

Carbon Monoxid.—This gas, which results from the combustion of high explosives, produces similar, though less intense, symptoms than the illuminating gas poisoning of civil life. Thus we find headache, asthenia, amnesia, delirium and syncope; symptoms which result from methemoglobinemia. Such cases are sometimes evacuated as "shock."

Chlorin.—The lesions and symptoms of chlorine poisoning closely resemble those of diphosgen but the immediate irritant effect upon the respiratory passages (larynx, bronchi, etc.) is much greater, although a much higher concentration is necessary to produce severe pulmonary edema. Thus violent and prolonged coughing occurs. Emphysematous changes, both pulmonary and subcutaneous are observed. The expectoration, which is copious, has a bloody, frothy character. Cyanosis is much more common than pallor. As a rule the onset of symptoms occurs promptly.

Nitrous Fumes (nitric oxid, nitrogen, peroxid).—These gases are liberated from burning cordite and may be encountered if an ammunition dump burns up. They also occur in mining operations if a blasting charge fails to detonate but burns slowly. In the latter instance the danger is a brief one as the gases are rapidly absorbed by moist earth. Although less toxic than chlorin in the production of pulmonary edema, the danger of nitrous fumes lies in their lack of odor and irritation. Delay in the onset of acute symptoms is very marked, and low concentrations of the gas, of which the subject is quite unconscious, may be fatal. If sufficient gas is present in the air to cause irritation of the respiratory passages the danger is great.

Chloropicrin.—This gas causes lachrymation, chest pain, abdominal discomfort and violent vomiting. Prolonged exposure to low concentrations causes serious toxic symptoms. It is chemically more stable than phosgen and cumulative in action, but in order to produce pulmonary edema, a higher concentration is required than is the case with phosgen.

Phenyl-carbylamin-chlorid.—The inhalation of this gas which has an offensive, mustard-like odor, produces immediate nausea, lachrymation, and later bronchitis. It does not rank high as a lung irritant.

In recent warfare gases were often mixed and hence it was often difficult to decide in mildly gassed men, with which type of intoxication one had to deal.

After Effects of "Gassing."—Information on this aspect of the subject is not complete as yet. The degree of permanent damage, even in those who have apparently completely recovered, will depend largely on the intensity of the initial inflammatory change in the bronchi. The pathological changes which occur in "gassed" cases are almost identical to those which are seen in influenza, both grossly and microscopically. In both there is inflammation of the bronchial mucosa, softening of the bronchial wall and a peribronchitis. The changes will vary in accordance with the severity of the infection in the case of influenza and the degree of concentration in the case of the irrespirable gases. In both instances the lungs in fatal cases often present the evidence of an intense hemorrhagic pneumonitis with or without definite areas of consolidation.

In the few gassed cases I have had the opportunity of studying with the X-rays, there has been a considerable increase in the trunk shadows and the evidence of fibrous changes about the bronchi. The roentgenogram is very similar to that seen in many influenza cases sometime after the attack (see Fig. 319). It is quite likely that in not a few instances this fibrosis will increase and lead to more or less extensive changes, especially in the lower lobes where the process seems to be more marked. In addition it is highly probable that bronchiectasis will be one of the ultimate effects in those cases in which the fibrosis of the lungs is at all extensive.

To what extent "gassed" individuals will become tuberculous is I think a matter of conjecture. Several cases I have seen were discharged from the army because they were believed to have had tuberculosis but in none of them did the condition exist. The cough, expectoration and signs at the bases of the lungs seemed to be due to a chronic bronchitis and peribronchitis. I should say that the chances of these individuals becoming tuberculous were no greater than others suffering from a similar pathological process but produced by other causes.

An interesting sequel to the inhalation of an irritant gas is bronchitis fibrosa obliterans. A few cases of this nature have been reported in civil practice.

Gases Encountered in Civil Life.—In civil life *nitrous and nitric acid fumes* are the ones which most frequently cause trouble. The inhalation of these fumes may occur in laboratories or factories in which considerable quantities of these gases are formed either during the course of some reaction or when a carboy of nitric acid is spilled. Wood[1] in reporting a personal observation with autopsy findings, has collected 9 similar cases. In addition he found in the literature references to a very considerable number in which respiratory symptoms were severe but in which the autopsy reports were incomplete or wanting. Exposure to the fumes may occur in a variety of ways and one or a number of individuals may be simultaneously affected. Pott[2] reports an instance in which thirty individuals were exposed to nitrogen tetroxid fumes formed by the action of acid phosphate on sodium nitrate in the preparation of a fertilizer. There were two deaths and in addition eight more individuals were confined to bed for a number of days with severe respiratory symptoms (dyspnea, cough and the expectoration of thick, yellow sputum containing blood). Hall and Cooper[3] report a similar experience

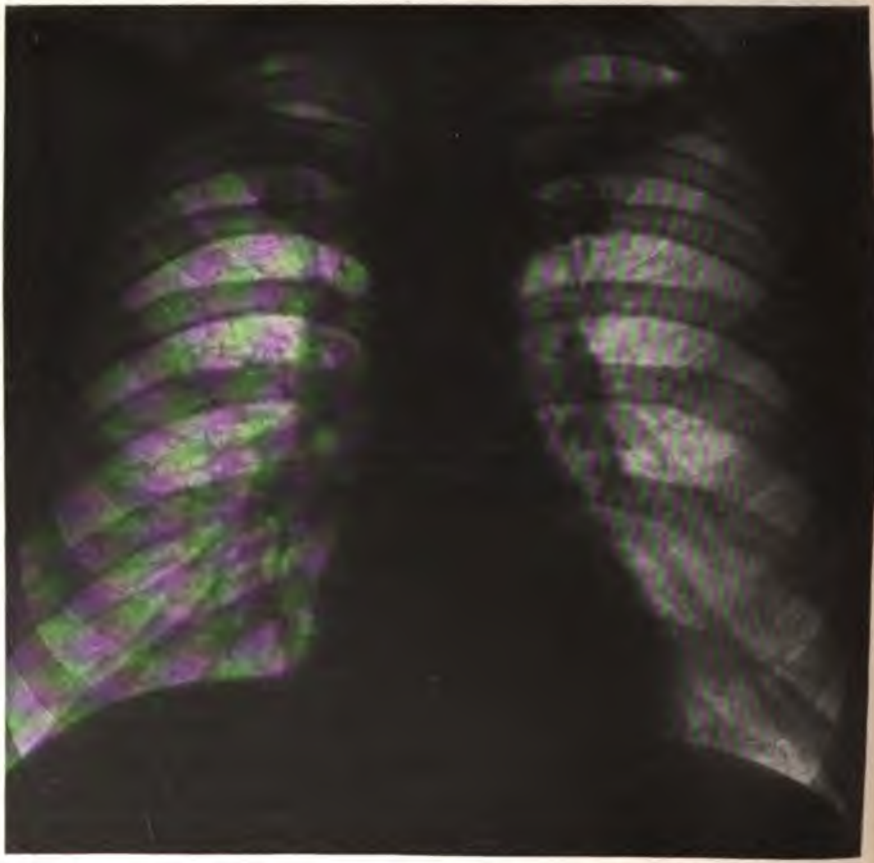

FIG. 319.—Radiogram showing after effects of "Gassing." Variety of gas unknown. Note marked increase in trunk shadows (peribronchitis).

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[1] *Trans. Assoc. Amer. Phys.*, 1912.
[2] *Deut. Med. Woch.*, 1884, x, 451.
[3] *Jour. Amer. Med. Assoc.*, 1905, xlv, 396.

which twenty people were exposed to nitric acid fumes. In this in-
ance a carboy of nitric acid was broken and in spreading over the floor
tacked some zinc plates and sawdust, starting a fire. All developed
arked respiratory symptoms and four died. Exposure to nitrous fumes
o occurs among those engaged in the manufacture of explosives and
ose exposed to the fumes which follow blasting. In mines when dyna-
ite, gelignite and nitro-explosive compounds are used the fumes
ven off from the explosives may travel to other portions of the mine
d affect the men working there or the men may return to the scene of
sting too soon after the explosion has occurred. Macaulay and
ine state that in the year 1904–1905 forty deaths were due to "gas-
g" in the South African mines and of this number seventeen were
sed by nitrous fumes. The diagnosis was confirmed by autopsy
half the cases.

In the case of most of the irrespirable gases the fumes are so noticeable
reason of their odor or their irritating properties that the workmen
ely warned as to their danger. It is the absence of these properties
makes the fumes generated by nitric acid so dangerous in industry
ay. The oxids of nitrogen are set free in large quantities in the
ing of sulphuric and nitric acids; in the nitration of cotton, in the
ing of trinitro-toluol, picric acid, etc., and also whenever metals are
ted with nitric acid. As there is nothing to warn the workman he
nues to work for a long time in air charged with these fumes. The
effect may be a slight feeling of suffocation, a little coughing and an
taste in the mouth. Any feeling of discomfort quickly passes off
reaching the fresh air. A few hours later, however, the patient
suddenly develop an attack of acute congestion and edema of the
s, preceded by an abundant expectoration of thick, yellow material.
ome instances these symptoms may be delayed as long as 24 to 48
s. In case the concentration of the fumes is great the symptoms
develop very quickly. These symptoms consist of a sense of op-
ion in the chest, dyspnea, cough, faintness and cyanosis. After
ing the fresh air, the patient may vomit and the respiratory symptoms
up. After an interval of 6 or 8 hours, the patient is suddenly seized
intense dyspnea and a sense of oppression in the chest, the eyes
de and a cold sweat appears. The attack is not unlike an asth-
seizure. In addition there are cyanosis and paroxysms of coughing
may last ten or fifteen minutes. Death occurs in consequence
na of the lungs which is usually preceded by the expectoration
y yellow fluid. In acute cases death may occur within 48 hours;
ally the patient may linger on for one or two weeks.

rin gas frequently constitutes an industrial hazard. The gas is
tant to the respiratory tract and men engaged in the manufacture
ing powder, for instance, can bear exposure to the fumes for not
n half an hour. Even then it is necessary to protect the eyes
gles and to cover the mouth with some material which will
he inhalation of the fumes. It is said, however, that chlorin
cquire considerable tolerance and can withstand gas in con-
that would produce glottic spasm in the untrained.

langerous gases often met in industry are bromin, hydrofluoric
hloric acids; nickel carbonyl, ferri carbonyl and sulphuretted

hydrogen gas. Nickel and ferri carbonyl are both very volatile liquids, the fumes of which are extremely poisonous.

Oliver states that taking chemical workers as a whole it may be said that the younger men are not an unhealthy class. The continued exposure to irritating gases, however, eventually renders them subject to bronchitis, asthma, and other respiratory troubles.

HYDATID DISEASE OF THE LUNG AND PLEURA

Hydatid disease of the different viscera is due to the ovum of the *Tœnia echinococcus* of the dog. In man the ovum develops into the larva—the hydatid—and persists in that state.

Etiology.—Hyatid disease has a fairly definite geographical distribution. The vast majority of cases have been reported from Iceland, Australia and the Argentine. Wherever man is brought into intimate contact with infected dogs the disease is relatively common. Dogs used for herding sheep are a common source of infection and this largely explains the frequency of the disease in Australia and the Argentine. In this country sporadic instances of the disease are occasionally encountered and at times an imported case is seen.

The abdominal viscera, particularly the liver, are the most frequent sites of the disease. Among 1863 cases of hydatid disease, the lung or pleura was involved in 153 or 8.3 per cent.[1] Thomas[2] collected 809 cases from the Australian Hospitals and of this number the lung or pleura was involved in 136, or 16.8 per cent. In a series of 952 cases reported from the Argentine by Vagas and Cranwell[3] the lung and pleura were involved in but 54 or 5.6 per cent.

The sexes are equally affected. As a rule the disease occurs among the lower class.

Morbid Anatomy.—The ovum of the tænia is eliminated with the dog's feces. Infection of human beings may be brought about by contaminated food or drinking water or by the hands becoming soiled in petting dogs. Direct infection of the lung through the inhalation of dust containing the ova was believed by Bird of Australia to be a possibility, but this theory has received no support.

The accepted teaching is that the ovum is taken into the stomach where the envelope is dissolved. The liberated ovum then bores through the wall of the stomach or intestines and in the majority of instances is carried by the portal vein to the liver. Thus in 1863 collected cases 953 or 51 per cent. the disease was localized in the liver. How the ovum reaches the lung is not clear. In many instances it apparently passes through the vessels of the diaphragm this assumption being strengthened by reason of the fact that the liver is so frequently involved and that in addition, the right lower lobe is the most common site in the lungs. Involvement of other portions of the respiratory tract probably results from the ovum entering a systemic vein and eventually reaching the lungs through the pulmonary arteries. Echinococcus infection of the lungs may be primary or secondary. When secondary the disease may extend from the liver or the infection may arise through metastasis from the liver or some other organ. Having become localized in the pulmonary or pleural tissues the ovum loses its hooks and is gradually transformed into a cyst. The cyst wall is composed of a stratified or laminated wall, the inner layer being granular or parenchymatous in character. From this parenchymatous layer brood capsules, scoleces and finally daughter cysts may develop.

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[1] Combined statistics of Davaine, Cobbold, Finsen and Neisser.

[2] "Hydatid Disease," 1894.

[3] *Revue de Chir.*, vol. xxiii, 970, 1901.

In the lungs hydatid disease may occur in the form of a single cyst which varies considerably in size; it may attain the dimensions of a fetal head. In some instances the cyst is multilocular and rarely numerous small cysts may be scattered throughout the lung. When the disease is located in the pleura it usually occurs in the form of a single cyst.

The contents of the cyst consist of a clear, transparent fluid containing the characteristic hooklets. In some instances the fluid may be sterile or as the result of secondary infection the fluid may contain pus.

The effect of the cyst is that of an irritant. The pulmonary tissue about it may become congested and later undergo fibroid changes. In cases of long standing the wall is apt to be very thick and fibrous. The cyst may rupture into one of the bronchi, into the pleural cavity or, rarely, into the pericardium. Rupture into a bronchus is the most usual termination. This may end in complete recovery but if the wall is thick secondary infection and gangrene may result. Rupture into the pleural cavity is usually followed by a fatal result.

Localization of the echinococcus in the pleura is not common and when the thoracic organs are involved it is the lungs that are usually affected. All statistics bear out the fact that the right lower lobe is involved far more often than any other portion of the lungs.

Symptoms.—The presence of a cyst in the lung or pleura may give rise to symptoms during the early stages when the growth is small or if it is centrally located and does not cause pressure symptoms. As long as the cyst remains intact constitutional symptoms are absent and the patient presents a healthy appearance. As the cyst increases in size, however, a dry, hacking cough usually occurs and this may be accompanied by mucoid expectoration.

The most important symptom is hemoptysis. This is the most frequent and the most striking symptom of the disease and occurs in the majority of cases. It may appear as an early manifestation; during the course of the disease; or coincidently with rupture of the cyst. In common with tuberculosis the first evidence of the disease may be blood spitting and as Dieulafoy has well expressed it, "the first cry of revolt on the part of the lung against the invader is perhaps a means of defense." In the early stages of the disease the hemorrhage may be slight but if associated with rupture of the cyst the bleeding is apt to be profuse.

Pain may or may not be present. If the pleura is not involved either primary or secondary pain is usually absent. There may be, however, a sense of weight or oppression in the chest.

Dyspnea is not apt to be a feature of the disease unless the cyst attains a large size. Fever is not apt to be present unless there has been a secondary infection of the cyst contents or an associated inflammation of the lungs, such as broncho- or croupous pneumonia.

Pressure symptoms will depend on the size and location of the growth. They are not common. The localization of the disease in the right lower lobe in a large proportion of the cases, precludes this possibilit When the disease is so situated as to cause pressure symptoms they a identical with those produced by tumors.

Rupture of the cyst occurs in about half of the cases and, as a rule, t rupture occurs into a bronchus. Rupture into a bronchus is attende by a violent paroxysmal cough, urgent dyspnea and pain or a sense o oppression in the chest. These symptoms are accompanied by the ex pectoration of a large quantity of clear transparent fluid. In some instances there may be, in addition, a brisk pulmonary hemorrhage. Examination of the expectorated fluid may show the presence of the hydatid hooklets or fragments of the cyst wall, the latter resembling the skins of grapes.

Rupture into a bronchus may be followed by an almost immediate fatal result. The evacuation of the cyst may be complete in a few days or it may be intermittent and extend over a period of weeks. If the cyst is old and the wall has become thickened secondary infection may occur. This gives rise to a purulent expectoration which gradually disappears. Gangrene of the cyst wall and surrounding tissues may occur.

Rupture into the pleural cavity is attended by the sudden onset of severe pain, dyspnea and signs of shock similar to those encountered in pneumothorax. Rupture into the pericardial sac produces pain in the chest and serious circulatory disturbances.

Physical Signs.—*Inspection.*—If the cyst is large and located in the pleura or near the periphery of the lung there may be bulging of the chest wall; after evacuation retraction may be noted. Restriction of motion is usually noted in that portion of the chest which overlies the cyst. Pressure signs are rare.

Palpation.—This may reveal slight degrees of diminished expansion. If the cyst is in contact with the chest wall, the tactile fremitus will be diminished.

Percussion.—If the cyst is in contact with the chest wall the percussion note over it is absolutely dull. The dulness may be modified, however, if a thin layer of pulmonary tissue intervenes. If the cyst is situated in the lower part of the chest, the dull percussion note may lead to a diagnosis of effusion. In the case of a cyst the upper level of dulness may be in front or in the axilla. In the case of an effusion the highest point of dulness is always posterior near the spine. After evacuation the percussion note over the site of the cyst may be tympanitic or amphoric in quality.

Auscultation.—Over the site of the cyst the breath and voice sounds are absent. After the cyst has become emptied there may be cavity signs: cavernous breathing, whispering pectoriloquy and metallic râles. The auscultatory signs surrounding the cyst will depend on whether inflammatory changes have taken place in the pulmonary tissue. Nothing abnormal may be heard or there may be râles and an alteration in the character of the breath sounds.

Diagnosis.—In countries where the disease is endemic, mistakes in diagnosis are less likely to occur than in those regions where an occasional sporadic case is seen. Prior to rupture of the cyst it is not possible to make an absolutely positive diagnosis. If in a suspected case the lesion is unilateral, basal and right-sided and in addition the X-ray examination shows a globular shadow, echinococcus disease is probably present.

After rupture of the cyst the diagnosis is much easier. The location of the lesion and the sudden expectoration of a large quantity of clear fluid s highly suggestive. The finding of the hydatid hooklets or fragments of the cyst wall is the only means of being absolutely certain of the diagnosis.

Echinococcus disease is most apt to be confused with tuberculosis, a pleural effusion or a thoracic tumor.

Tuberculosis is either confined to the summit of the lung or if the disease involves the entire lung the physical signs are always most marked at the apex. Although hydatid disease may involve the upper portion of the lung its usual location is at the right base. Tuberculosis nearly always causes shrinkage of the chest wall while a cyst produces bulging. A hydatid cyst may give rise to marked dulness anteriorly with a normal note posteriorly or the dulness may extend across the middle line.

Active tuberculosis is always attended by constitutional symptoms particularly fever and loss of weight while a hydatid cyst rarely affects the general health unless secondary infection with the pus cocci or gangrene develops after rupture.

In the case of a large basal cyst the condition may simulate a *pleural effusion*. In the latter the line of dulness is always higher posteriorly near the spine, there may be movable dulness and over the upper limits of the effusion egophony is commonly heard. Pain or tenderness on pressure is common in pleurisy and is often absent or very slight in hydatid cyst. The contour of the shadow in the X-ray plate may serve to distinguish the two conditions, that of the cyst being globular in outline.

If pressure symptoms are present, the differentiation between a hydatid cyst and a *malignant tumor* may be difficult. In the former constitutional symptoms are slight or absent while in the case of the latter the evidences of a rapid decline sooner or later manifest themselves.

PULMONARY DISTOMATOSIS

Etiology.—This affection is known also as lung fluke disease, parasitic hemoptysis, endemic hemoptysis and paragonimiasis. The disease is deserving of some attention, for, although the United States are not included in its geographical distribution, isolated cases are apt to be encountered at any time, especially among those who have resided in the far East. The disease occurs in Japan, China, and Korea. Among the inhabitants of the Island of Formosa endemic hemoptysis is very prevalent and in certain districts as high as 15 per cent. of the population is said to be infected. It has also been noted as occurring in the Philippine Islands. A few cases have been observed in this country. The disease also has been encountered in the tiger, dog, cat, and in swine.

The exciting cause of pulmonary distomatosis is a trematode worm, the lung fluke, known as *Paragonimus Westermannii*. The worm is almond-shaped and in the fresh state of a pinkish or reddish-brown color. The eggs are golden yellow in color and larger than the eggs of other parasites common to man. They may be seen with the unaided eye as brownish specks if a small portion of sputum containing them is pressed between a cover slip and slide and held up to the light.

The life history of the first and final stages only is known. Manson found that if the sputum containing the eggs is mixed with water and the water changed occasionally, after some weeks, more or fewer according to temperature, a ciliated embryo will develop in each egg. In a short time the embryo emerges from the egg and swims about vigorously for some hours. It thus seems probable that the sputum cast on the ground is washed down by the rain or otherwise and so gets into wells or pools of water. Beyond this we have no positive knowledge but Manson assumes that the embryo gets into a small mollusc, in which it undergoes the evolutionary changes characteristic of other distomes. When these are completed it is carried in the water or by some water plant, back to man again.

Nakagawa[1] has found encysted larvæ in fresh-water crabs. The encysted larvæ were fed to puppies. Examination after death revealed a number of cysts in the lungs. Further study showed that the encysted larvæ after they have been taken into the alimentary tract of the host escape from the cysts, penetrate the intestinal wall near the jejunum and escape into the abdominal cavity. They then pass through the diaphragm into the thoracic cavity, scatter over the pleura and finally gain entrance into the pulmonary tissues. Having entered the parenchyma of the lung a cyst is formed and the worm becomes full-grown. Although the parasites may become fixed in organs other than the lungs the latter seem to be the most favorable place for their development.

Morbid Anatomy.—The lungs of individuals who have become the victims of this parasite contain a varying number of cysts or burrows which harbor the worm. These cysts are commonly about the size of a hazelnut but they may be as large or even larger than a walnut. They may be located at any point within the parenchyma of the lung but are most frequently found toward the periphery of the organ. If located just beneath the pleura they may project as hemispherical nodules of a bluish-gray color. The frequency with which the cysts occur near the periphery of the lungs leads to secondary inflammation of the pleura and as a result adhesions are common.

According to Manson these so-called burrows are little tumors or thickenings produced by inflammatory exudate in the parenchyma of the lung and are riddled with small passages or burrows in which the parasites lie. Each cyst or burrow contains one or more worms and in addition a reddish, dirty brown, mucosanguineous material. The cavities communicate with the bronchi by one or more passages and at times neighboring cysts communicate with each other by tortuous channels. Again adjacent cysts may rupture into each other forming large irregula cavities.

Although there is a direct passage from the burrow containing th worm to the outside by way of the bronchi, the worms themselves ar rarely coughed up. According to the size and number of the cysts, certain amount of the rusty, ova-bearing sputum is expectorated o swallowed. The extent of the infection has a bearing on the severit of the symptoms. It may be so slight that the patient experiences n difficulty other than the expectoration of a little rusty colored sputum on the other hand, the patient may be subject to recurring attacks o profuse hemoptysis.

While the lung is the normal habitat of the parasite, it may b und in other parts of the body such as the scrotum, liver, peritoneum, iaphragm, lymph nodes, and especially the brain. In the latter situaon it may produce the symptoms of Jacksonian epilepsy or brain umor. Japanese observers have reported instances of brain infection which cysts containing both the ova and worms occurred.

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[1] *Jour. Infect. Dis.*, xvii, No. 2, p. 131.

Symptoms.—The disease is usually insidious in its onset and as rdinarily seen, not attended with marked constitutional symptoms or y emaciation. The distinctive features of pulmonary distomatosis re cough, which is usually worse on arising in the morning, the expectoraon of rusty, brown or dark red sputum and the occurrence of recurring ttacks of hemoptysis. Owing to the close proximity of the "burrows" o the visceral layer of the pleura, inflammatory changes in the latter re common, thus giving rise to varying degrees of chest pain. In common with other chronic pulmonary lesions patients who are subject to his infection are apt to suffer from dyspnea, and mild asthmatic attacks. If the attacks of hemoptysis are frequent, the patient may become anemic and in such cases functional murmurs are heard. As a rule there is no fever. The severity of the symptoms depend largely on the amount of the pulmonary damage. The disease may be so limited in extent as to cause no inconvenience other than the slight cough and expectoration. In other instances, the patient is subject to frequent attacks of profuse bleeding and a gradual deterioration in health.

In the great majority of instances the lungs alone are involved, but as already pointed out, other portions of the body may be involved either primarily or as a complication of the pulmonary infection. Next to the lungs the brain seems to be the most vulnerable point. Involvement of the brain gives rise to a variety of nervous symptoms: epilepsy, hemiplegia, paresis of the extremities, headache, vertigo, etc.

Physical Signs.—In common with other pulmonary infections which simulate tuberculosis there is nothing distinctive in the physical signs which will serve to differentiate the two conditions. Physical signs may be entirely wanting if the lesion is small or deep-seated. In cases with extensive involvement of the pleura there may be marked retraction of the chest wall and a very limited respiratory movement. The lesion may be single and circumscribed or there may be a number of such areas over which the percussion note is impaired and the breath sound suppressed or broncho-vesicular in character. Râles may be present also. Signs of cavity formation are not commonly encountered.

Diagnosis.—Inasmuch as cough, blood-streaked sputum or a frank hemoptysis and chest pain are the principal symptoms of pulmonary distomatosis, the disease must be distinguished from other conditions in which the same symptoms occur. It is evident that the disease which most commonly gives rise to such symptoms is *tuberculosis*. So far as the symptoms are concerned no distinction between the two is possible and if the lung fluke is located in the pulmonary apices the confusion is increased. A very important point is the question of whether the individual has lived in the endemic zone. The diagnosis must rest on the microscopic examination of the sputum and this is easily made from the presence of the eggs and the absence of tubercle bacilli. Both tuberculosis and distomatosis may occur in the same individual. In suspected cases in which it is impossible to obtain the sputum the stools should be examined for the ova. It is said that the presence of Charcot-Leyden crystals in the sputum is strongly suggestive of distomatosis in those living in infected regions and should lead to a careful search for the eggs.

SYPHILIS OF THE RESPIRATORY TRACT

LARYNX

Syphilis may attack the larynx both as an early and a late manifestation of the disease but is far more common in the so-called secondary stage. Indeed a very large proportion of individuals suffering from acquired syphilis manifest symptoms of laryngeal trouble, usually, however, of a transient nature. Any portion of the larynx may be involved —the epiglottis, the vocal cords or the body of the larynx.

Examination of the larynx during the active stage of syphilis will often show the presence of catarrhal changes, congestion of the mucous membrane, mucous patches or slight erosions. The catarrhal inflammation or congestion often becomes apparent at the time the cutaneous eruption first makes its appearance. In other instances the laryngeal lesion develops later. Mucous patches are much less common. They may form superficial erosions or in severe cases an ulcer may be formed. Edema of the laryngeal tissues may accompany any of these changes. Unless the individual is cured there is a tendency for the laryngeal lesions to recur.

Tertiary lesions of the larynx are relatively rare and usually appear very late. A gumma varying in size from a pinhead to a hazelnut, is usually the first manifestation. The gumma may break down forming a deep ulcer which in the process of healing may produce serious deformities as the result of retraction of the scar tissue. Partial or even complete laryngeal stenosis may be produced. In other instances the tertiary disease attacks the cartilaginous portion of the larynx. The disease may extend and by invading the tissues about the larynx produce an external fistula.

Symmers[1] in a study of 4880 autopsy protocols found the anatomic confirmation of the existence of syphilis in 314 cases, or 6.5 per cent. The larynx was involved in 12. In 5 there were cicatricial lesions; in 5 ulcerative lesions, in 1 a gumma and in 1 a leukoplacia of the epiglottis. In addition there were 17 cases in which the epiglottis was deformed in association with syphilitic changes at the base of the tongue.

In the *secondary stage* the most constant, and often the only symptom, is hoarseness. A cough is more often absent than present. Complete aphonia sometimes occurs. Rarely marked difficulty in breathing occurs as the result of edema of the larynx.

In the *tertiary stage* the trouble usually first manifests itself by hoarseness which may develop into complete loss of the voice. If cicatricial changes occur, the difficulty in breathing becomes progressively worse. Sudden closure of the laryngeal opening may occur. The difficulty in breathing is both inspiratory and expiratory and is accompanied by more or less stridor. If the ulcerative process involves the epiglottis or the arytenoids the patient may experience pain on swallowing.

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[1] *Jour. Am. Med. Assoc.*, May 6, 1916.

TRACHEA AND BRONCHI

Morbid Anatomy.—Involvement of the trachea alone is rare. In 03, Conner[1] collected 128 cases of syphilis of the trachea and bronchi. ymmers[2] found 4 instances, among 4880 autopsies, in which the lesion as limited to the trachea; and Lord[3] states that 2 instances of syphilitic lceration of the trachea were found among 3000 autopsies at the Massahusetts General Hospital.

Lesions in the trachea are present, as a rule, in the upper or lower hird, especially at the bifurcation. Involvement of the lower portion f the trachea and the main bronchi is not uncommon. One or both of he primary divisions of the bronchi may be involved.

The tertiary lesions of the trachea and bronchi are practically always ate manifestations and may be due to hereditary as well as the acquired orm of the disease.

The mucous membranes of the trachea and large bronchi may be the eat of catarrhal change during the secondary stage. This change may be the result of an increased susceptibility to "colds" during the acute stage of syphilis or, as in the case of the larynx, to the presence of mucous patches.

As a tertiary manifestation syphilis may occur in the trachea or large bronchi as a gummatous infiltration. At first this causes considerable swelling. The gumma may become absorbed leaving as evidence of its presence a scar or it may break down and produce an ulcer and this in turn may heal and become a scar. A syphilitic ulcer in the lower portion of the trachea or in one of the primary bronchi may perforate or extend into the surrounding mediastinal tissues, the esophagus or the pulmonary artery. Occasionally the cartilaginous structures become necrosed and either may be entirely absorbed or small pieces of cartilage may slough off and be expectorated.

When healing follows either a gumma or an ulcer, the resulting scar tissue usually contracts and often produces marked deformity or even complete stenosis. At the point of the scar the lumen is narrowed by an annular or membranous stricture or strictures. Dilatation of the trachea or bronchi may occur above and below the constricted point.

Symptoms.—During the secondary stage of syphilis involvement of the trachea may manifest itself as a simple trachitis. There is cough, expectoration and some substernal soreness.

As a late manifestation the first evidence of trouble is usually a cough, which at first is dry and unproductive; later it is accompanied by mucoid or mucopurulent sputum. If the disease occurs in the form of a gumma which becomes absorbed, the symptoms rarely consist of anything more than a cough with or without the presence of sputum. If, however, an ulcer forms the sputum is often blood-streaked and a frank hemoptysis is not unusual. A few instances have been recorded in which a fatal hemorrhage followed perforation of an ulcer into the pulmonary artery. Substernal pain may occur and in some instances there is also dysphagia.

With the formation of a stricture the symptoms of stenosis are added to the picture. As soon as the stricture begins to offer obstruction to the ingress and the egress of the air, the patient begins to suffer from dyspnea which is more or less marked, depending on the degree of the stenosis. The dyspnea may be persistent or it may occur in paroxysms. Suffocative attacks may come on very suddenly; sometimes without apparent cause, in other instances as the result of sudden exertion. The difficulty in breathing is both inspiratory and expiratory and is usually attended by marked stridor. During the attack the patient becomes cyanosed, the pulse becomes weak and there may be unconsciousness.

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[1] *Am. Jour. Med. Sc.*, vol. cxxvi, p. 57.

[2] *Loc. cit.*

[3] "Diseases of the Bronchi, Lungs and Pleura," 1915.

Physical Signs.—The respiratory movements of the chest are diminished, the breath sounds are feeble and the tissues at the root of the neck, the lower intercostal spaces and the epigastrium show inspiratory retraction. Rarely one of the main bronchi may be stenosed. This is to be suspected if there is unilateral diminution of the respiratory excursion and suppression of the auscultatory phenomena. There may be present also, over the site of the stricture, a whistling quality of the respiratory sounds.

In Conner's[1] series death occurred in eleven instances during a suffocative attack.

Diagnosis.—Prior to the formation of a stricture tuberculosis is most apt to be the diagnosis owing to the cough, mucopurulent sputum and the appearance of blood. The persistent absence of tubercle bacilli and of abnormal physical signs in the lungs renders the correctness of such a diagnosis doubtful. Syphilis is always to be thought of, especially if there is a history of that disease, the presence of syphilitic stigmata elsewhere and the presence of a positive Wassermann test.

If stenosis has taken place the obstruction may be due to a syphilitic stricture, to a foreign body or to the presence of a tumor, aneurism or enlarged lymph node. The X-rays and the use of the bronchoscope should always be employed in such cases.

THE LUNGS

Syphilis of the lungs has always been looked upon as a rare condition. The diagnosis is not often made during life. If the lesions have become healed the resulting scar tissue differs in no particular from that produced by a non-specific inflammation, hence, it is difficult to prove, even after death, whether syphilis has been present or not.

It is quite likely that pulmonary syphilis will become less of a rarity now that we know the exciting cause. Clinically the most important addition to our knowledge has been the introduction of the Wassermann test.

Morbid Anatomy.—The incidence of pulmonary syphilis is based entirely on the finding of the lesions in the lung which are unmistakably luetic in character. As already stated, extensive fibrosis of the lung or the presence of localized areas of scar tissue may or may not be syphilitic; as a rule such lesions are rarely classed as such. Warthin[2] has reported recently a histological study of the pancreas in individuals who had had syphilis. He was able to demonstrate that the pancreas showed unmatiskable evidences of the presence of areas, of various sizes, which were due to syphilitic changes. In many instances the presence of spirochetes could be demonstrated also. It is quite possible that if the lungs are subjected to a similar study syphilis will be found to be a more frequent invader than, at present, is believed to be the case.

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[1] *Loc. cit.*

[2] *Trans. Assoc. Am. Phys.*, 1916.

Based on present standards, syphilis of the lungs in adults is a rare condition. This is shown by the following figures: Fowler was able to find but 12 examples of the disease, 2 of which were doubtful, in the museums of the London hospitals. Among 2800 autopsies at the Johns Hopkins Hospital there were 12 cases with syphilitic disease of the lungs and 8 of these were due to the congenital form (Osler); among 3000 autopsies at the Massachusetts General Hospital but 1 case showing indurative pneumonia, with cavity formation, was found (Lord); in a study of 4880 autopsy protocols, 314 of which showed lesions due to syphilis, Symmers[1] found the lungs involved in 12 cases and the presence of syphilitic pleural scars in 2 more.

Congenital Syphilis.—The only type of pulmonary syphilis about which there is a general agreement is that seen in new-born syphilitic children. In this form of the disease gummata may occur, but as a rule are rarely seen. The typical lesion is that known as white pneumonia. The lung of a syphilitic fetus or infant is much increased in size and may be marked by the ribs. It presents on section a dry, smooth surface, grayish or yellowish-white color, resembling very closely the cut surface of the pancreas. The areas of consolidation may occur in scattered patches throughout both lungs or they involve an entire lobe.

The chief changes are cellular infiltration involving the alveolar walls and proliferation of the interlobular connective tissue thus giving rise to an interstitial pneumonia. Stained sections from the lungs of syphilitic children show an enormous number of the spirochatæ of syphilis.

For the most part this type of pulmonary syphilis is of pathological interest only.

Acquired Syphilis.—In acquired syphilis three types of the disease are encountered in the lung, namely, gummata, fibroid changes and areas of consolidation and catarrh.

1. *Gummata.*—When the disease occurs in the lungs in this form the gummata are apt to be quite numerous. They vary greatly in size ranging from that of a millet seed to a hen's egg. In the early stages of their development gummata are grayish red or grayish white in color, somewhat translucent, and are surrounded by an area of congestion. Later they become opaque and somewhat caseous. In addition they become walled off by a thick fibrous envelope. Occasionally a gumma may caseate and empty into a bronchus, thus producing a pulmonary cavity. This is a very rare manifestation of the disease known as syphilitic phthisis. Eventually the gummata may become entirely healed, leaving behind them nothing but a tough mass of puckered scar tissue cannot be distinguished from similar scars produced by tubercles bscess.

gummata may occur in any portion of lung but are usually to be about the hilus and in the lower lobes.

2. *Fibroid Induration.*—This is a much more frequent manifestation n the formation of gummata although the latter may also occur in association with the fibroid changes. Now that the exciting cause of

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[1] *Loc. cit.*

syphilis is known the presclerotic changes are better understood. Following the lodgment of spirochatæ in the lung an inflammatory reaction sets in. This takes the form of an extensive cellular infiltration together with an overgrowth of the connective tissue in the interlobular septa and proliferation and desquamation of the alveolar epithelium. The alveolar walls become greatly thickened as do also the blood-vessels. Gradually the entire area is converted into dense fibrous tissue. When this stage is reached, the clinical picture is that of pulmonary fibrosis or bronchiectasis. How frequently syphilis is the exciting cause of a diffuse pulmonary fibrosis is problematical.

The indurative changes may originate at the hilus of the lung and extend outward along the bronchi and blood-vessels or the starting point may be the pleura and interlobar septa. The process is usually unilateral and at most involves only a portion of one lobe; if several lobes are implicated, it is the portions which adjoin the root of the lung.

3. *Consolidation and Catarrh.*—In addition to the types just mentioned a focal form is described in which the lesion consists of an area of consolidation and catarrh. It may be situated around the root of the lung or it may occur at one apex. The general opinion has been that the apices are rarely involved, but it would be rather surprising, in view of the wonderfully diverse forms in which the localization of syphilis manifests itself, if the upper portions of the lung should escape. Lewis and I[1] have reported, from the Phipps Institute, six cases in which the physical signs pointed to a focal lesion in the apex and in which the evidence seemed to be conclusive that a latent syphilitic process was present. During the past two or three years I have seen additional cases of this nature. Lisser[2] has reported 7 cases and emphasizes the importance of keeping syphilis in mind in cases having many of the symptoms and signs of pulmonary tuberculosis but no tubercle bacilli in the sputum.

In the secondary stage of syphilis symptoms and physical signs pointing to a catarrhal condition of one apex are occasionally encountered. Dunham[3] has referred to several cases of this nature. He believes the changes are analogous to those occurring in the larynx and trachea during the secondary stage.

Although the presence of spirochetes has been demonstrated in many of the organs of the body the lungs have not been studied very thoroughly in this respect. Two specimens have been shown before the Pathological Society of Philadelphia in which these organisms were found.

Symptoms.—The symptoms which have been noted as occurring in individuals suffering from syphilitic invasion of the lungs are not distinctive. They are such as may occur in any inflammatory process which involves the pulmonary tissues. In addition the symptoms will vary accordingly as the patient is seen in the active stage of the process or during the stage when chronic fibroid changes have developed.

The following pulmonary symptoms are usually present: Cough, which may be dry and unproductive or accompanied by a moderate amount of expectoration which is yellowish or greenish in color. Hemoptysis is not common although in one case which I had under observation ere were frequent attacks of blood spitting. Dyspnea is often present id may be very marked in those cases in which there are extensive roid changes. In some instances it is asthmatic in character. Hoarse-ss may be a prominent symptom as the result of changes in the larynx.

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[1] *Amer. Jour. Med. Sc.*, August, 1915.
[2] *Amer. Jour. Med. Sc.*, March, 1918.
[3] Kelly's Stereo Clinic, Section xxxvii.

If the case is seen after the fibroid induration has existed for some me the clinical picture is that of cirrhosis of the lung with or without latation of the bronchi.

Some years ago E. G. Janeway[1] emphasized the fact that when the scera become involved during the tertiary stage of the disease, a moder-e amount of fever is usually present, emaciation is marked and night veats are of frequent occurrence. These symptoms are often associated ith pain in the right side due to a syphilitic perihepatitis. Further-iore, one is apt to find, on careful examination, some other noticeable vidence of syphilis, such as an indurated testicle or enlargement and enderness of the ribs or clavicles at the sternal attachments.

Warthin[2] has shown that next to the aorta and heart the testes are he most frequent sites of a syphilitic infection. In a pathological study f 36 cases of syphilis occurring in males, the majority of which were tent and unrecognized during life, an orchitis fibrosa was present in 31. mong 171 male subjects of late acquired syphilis Symmers[3] found a hronic interstitial orchitis in 67 or 39 per cent. He believes that the liminished size of the testicle and its increased consistency furnish aluable evidence both in the clinical and anatomical diagnosis of yphilis.

In the case of women a history of miscarriages is extremely significant.

Physical Signs.—If the syphilitic manifestation is limited to the arynx, trachea or main bronchi, the diagnosis is a matter of direct inspec-ion by means of the laryngoscope or bronchoscope. Unless the examiner as had special training in this work the case should be referred for an xpert opinion. Involvement of the smaller bronchi or parenchyma f the lung does not produce physical signs, which are indicative of yphilis. In the vast majority of cases the primary diagnosis is that of uberculosis, either incipient or advanced, depending on whether there is focal lesion, or a diffuse fibroid process. The former may occur about he root of the lung, or at the apices. When an apex is affected the signs liffer in no particular from those encountered in incipient tuberculosis. n the fibroid form the condition is usually mistaken for fibroid phthisis r bronchiectasis. In very rare instances pulmonary gummata may reak down and empty into a bronchus, thus forming a cavity. In uch instances the physical signs would be identical with those encoun-ered in tuberculosis.

The diagnosis of syphilis of the lung is almost invariably made by a rocess of exclusion. An individual who has complained of cough, with r without expectoration, for several months, who has slight fever and nalaise and has suffered from some loss of weight, is almost certain to e regarded as being tuberculous. Pulmonary signs may be wanting or nay be present at one or the other apex. If the physical signs are located t the base or root of the lung one's suspicions should be aroused. As a eneral rule the true nature of the trouble escapes detection and is recog-ized only when the patient fails to improve or there develops some other evidence of the disease such as an orchitis or bone lesion. Now that the Wassermann test is available it should always be applied in suspicious cases. The therapeutic test is also of service in determining the true nature of the trouble. Following the use of salvarsan or mixed treatment the physical signs and symptoms show decided improvement. Inasmuch as iodide of potassium has a marked tendency to cause a breaking down of tuberculous lesions in the lung, the drug should never be administered until every means has been employed to exclude tuberculosis.

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[1] *Trans. Assoc. Am. Phys.*, 1898.

[2] *Loc. cit.*, 1914.

[3] *Loc. cit.*

The following cases will serve to indicate the various manifestations of pulmonary syphilis:

Case I (Phipps Institute No. 9485).—Female, aged 28, first came to the Phipps Institute June 10, 1911. She gave a history of having had repeated attacks of blood spitting for five years. Her first pregnancy resulted in a miscarriage. During the past year she had had a cough, considerable greenish expectoration, night sweats and had lost during the past two years 30 pounds. She also complained of pain over the base of the right lung (perihepatitis). There was a slight elevation of temperature, 99° to 100°F. Examination of right lung showed some flattening beneath right clavicle, diminished expansion, impairment of the percussion note and broncho-vesicular breathing. She was sent to a sanatorium and remained away several months. Although she gained 16 pounds in weight, the pulmonary symptoms remained unchanged.

On her return to the Institute her sputum was repeatedly examined for tubercle bacilli as well as other organisms but was always found to be negative. The Wassermann test was strongly positive. On April 27, 1912, was given salvarsan and afterwards put on mixed treatment. Three Wassermann tests within the next six months were negative. Guinea pigs inoculated with her sputum were killed and found to be free from tuberculosis. The pulmonary symptoms steadily improved. During the past year she became pregnant and recently was confined. At the present time, February 1, 1916, she weighs 188 pounds and is apparently well in every way.

Case II (Phipps Institute, No. 13391).—Female, aged 34 years, first visited the Phipps Institute August 20, 1914. Her family history was negative. She had been married eleven years and had had three miscarriages. No living children. There was no history of an initial lesion nor of secondaries. She came to the dispensary because of a cough, greenish-colored expectoration, night sweats and some loss in weight. In addition she complained of precordial pain, which had been present intermittently for six years. For four years she had suffered from attacks of burning and tingling in the legs.

Examination.—Some flattening beneath the right clavicle, impairment of the percussion note, and broncho-vesicular breathing. Heart was negative. The reflexes were exaggerated.

During the first six months she was under observation the night sweats, morning cough, and expectoration persisted, and the sputum was blood-tinged on one occasion. There was also a slight rise in temperature. The sputum was negative for tubercle bacilli. In February, 1915, she first complained of nocturnal headache. This gradually became worse, and finally was so severe as to interfere with her sleep. The Wassermann test was strongly positive. Under mixed treatment the headache was relieved. Later, following the administration of salvarsan, the cough ceased and the amount of expectoration was greatly reduced.

Case III.—A male aged 30 was admitted to the White Haven Sanatorium several years ago with a history of having been ill for some months. He had lost about 25 pounds, had a cough, expectoration, slight fever and marked hoarseness. Examination of the lungs showed impairment, feeble breath sounds and a few fine râles at the left apex. The case seemed to be clearly one of pulmonary and laryngeal tuberculosis. Two weeks after his admission the trouble in his larynx was found to be specific in nature. Under mixed treatment he made a complete recovery, the pulmonary signs entirely disappearing.

Several cases have been sent to White Haven because of marked hoarseness which on examination proved to be syphilitic in origin. With the exception of the case just mentioned pulmonary signs were wanting. These cases serve to emphasize the necessity of a competent laryngological examination, as delay in instituting antisyphilitic treatment may lead to serious and permanent damage of the larynx.

Case IV.—A middle-aged woman began to suffer from ill health about three years prior to coming under observation. She had lost weight, had a persistent cough with expectoration and in addition suffered greatly from dyspnea. At times she had moderately severe asthmatic attacks. Examination of her chest showed restriction of motion at the right base; the percussion note was dull, especially anteriorly at the base, and the breath sounds were broncho-vesicular in type. She had been under treatment during most of this period for tuberculosis but with no improvement. Persistent absence of tubercle bacilli lead to a Wassermann test which was positive. Several injections of salvarsan brought about some improvement in the symptoms but the physical signs remained unchanged. In this case the pulmonary process had probably advanced to permanent fibrosis.

Case V (Phipps Institute Case No. 1628).—A female, aged 52. Admitted to the Phipps Institute in 1903. She gave a definite history of having acquired syphilis at the age of 15. There were present interstitial keratitis, perforation of the palate, and numerous deep scars scattered over the body. The present illness began five years prior to admission with cough and pain in the right side. Two years later she had a hemoptysis and during the three following years had had over 30 such attacks. On admission she had cough, greenish expectoration, night sweats, dyspnea and some loss in weight. The sputum has been examined repeatedly for tubercle bacilli, as well as other organisms, but always with negative results.

Examination of the chest showed restriction of motion on right side, most marked at the base, dulness up to the angle of the scapula, distant breath sounds and fine cracking râles. At the angle of the scapula there was a small area of tympany on percussion, whispering pectoriloquy and bronchial breathing.

This woman has been seen from time to time during the past twelve years. The symptoms, including an occasional hemoptysis, have remained unchanged. It seems probable that in this case the pulmonary process (gumma or diffuse fibroid induration) has ended in a fibrosis of the right lower lobe and that, in addition, the bronchi have become dilated. A similar case has been reported by Osler.[1]

Diagnosis.—The most frequent error is that of mistaking pulmonary syphilis for tuberculosis. The symptoms are often identical. If the physical signs are elicited about the hilus or at the base of the lung, tuberculosis, almost invariably, can be ruled out. If, however, the lesion is located in the apex of the lung syphilis may simulate tuberculosis so closely as to deceive us entirely. To establish a diagnosis of syphilis the sputum must be negative repeatedly for tubercle bacilli, or other organisms which produce lesions of a similar nature; other syphilitic stigmata must be present, such as a bone lesion, keratitis, indurated testicle, etc.; and the Wassermann test must be positive. The results of antisyphilitic treatment also furnish valuable evidence. Tuberculosis, for instance, will not be improved either by the use of salvarsan or of mercury and the iodides. Syphilis on the other hand is markedly improved or cured by the use of these drugs unless permanent fibroid changes have taken place.

Syphilis and Tuberculosis.—The prevalence of both these diseases makes it inevitable that many cases are encountered in which the same individual is suffering from both infections.

In one who has been the victim of syphilis for many years the occurrence of active tuberculosis may be brought about by a lowering of
stance as a result of the primary infection. The course of the
culosis does not differ, however, from that occurring in non-syphilitic
duals.

the other hand, if both diseases develop simultaneously or within
t time of each other, the patient is apt to be overwhelmed. The

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[1] Power and Murphy: "System of Syphilis," vol. iii, p. 24.

tuberculous infection is especially apt to become extremely active and to pursue a rapid and acute course. There are exceptions, of course, but it is generally recognized that the association of the two diseases in an active form, is a particularly vicious one and that the tuberculosis is rarely arrested in such cases. Distressing examples of this are often seen in young prostitutes who have developed tuberculosis in the early stages of the secondary period of syphilis and in whom the tuberculosis runs a very rapid course.

INTRATHORACIC TUMORS

For clinical purposes it is more convenient to include all tumors arising within the thorax under one general heading. This seems best because of the fact that the symptoms to which they give rise are in the majority of cases similar; although here and there a case is encountered in which the symptoms and physical phenomena point definitely to the growth being limited to a bronchus, the lung, the pleura or some portion of the mediastinum. Furthermore, the morbid anatomy of the tumors and the conditions under which they invade the thoracic viscera have no striking differences.

Etiology.—The malignant tumors may be primary or secondary, the latter being by far the most common. Malignant growths may occur at any age. Up to the middle period of life the tumor is almost invariably sarcomatous in nature; the younger the subject the more certainly is this true. Beyond middle life the growth may be a carcinoma or a sarcoma, usually the former. Endotheliomas, tumors which histologically occupy a position midway between carcinoma and sarcoma, may occur at any age. Primary malignant growths affecting the organs of the thorax occur more frequently among males than females. Owing to the greater frequency of cancer among women metastatic growths are more frequently encountered in this sex. In a series of 71 cases of metastatic malignant growth studied by Moore and Carman[1] 20 were secondary to cancer of the breast while in but 2 cases was the primary growth in the uterus. Trauma of the chest wall has been cited as an occasional cause of malignant disease arising within the thorax.

Ancke[2] has reported the interesting fact that the Cobalt miners of the Schneeberg district in the Saxon Voigtland are very liable to primary sarcoma of the lung. Whether it is due to irritation from dust (Adami) or whether the endemic occurrence points to an infectious origin (Strümpell) is uncertain.

Primary cancer of the lung is generally looked upon as of relatively rare occurrence. According to Sailer and Torrey[3] this belief is not well founded. By combining various statistics they were able to collect 87,451 autopsies among which 130 primary carcinomas of the lung were found; that is, of 100,000 deaths, 156 are caused by cancer primary in the lung. McMahon and Carman[4] place the number of authentic cases of primary carcinomas at 428.

Primary carcinoma of the large bronchi is not common. Weller[5] has collected 90 cases.

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[1] *Amer. Jour. of Roentgenology*, March, 1916.
[2] *Dissert.*, Münch., 1884.
[3] *Penna. Med. Jour.*, April, 1913.
[4] *Amer. Jour. Med. Sc.*, Jan., 1918.
[5] *Arch. Int. Med.*, March, 1913.

Primary involvement of the pleura by a carcinoma is rare, there being not more than 40 or 50 cases on record. It is possible as Lord suggests, that instances of the disease have not infrequently escaped notice because of the readiness with which it may be confused with thickening of the pleura due to a chronic inflammatory process.

Primary sarcoma of the lung is much less common than carcinoma. Passler[1] found among 1000 cases of malignant disease 16 cases of primary carcinoma of the lung and 4 of primary sarcoma; Seydel[2] in an analysis of 10,829 autopsies found 20 primary carcinomas and 8 sarcomas. Rolleston and Trevor[3] in 3983 autopsies found three primary sarcomas and no carcinomas of the lung. Lord[4] has collected 42 cases in which the tumor was an undoubted or probable primary sarcoma.

Primary sarcoma of the bronchi and pleura is even rarer than primary carcinoma. Bernard[5] has collected 24 instances of primary sarcoma of the pleura.

The lungs may be invaded secondarily by a malignant growth by direct extension from adjacent structures or by metastasis from some distant organ. If the growth is a carcinoma the initial focus may be one of the abdominal viscera such as the stomach, liver or pancreas, or the primary growth may be in the prostate. Metastasis may take place by way of the blood stream or the lymphatics. Direct extension of the disease may occur when the primary focus is in the mammary gland, esophagus or thyroid gland. According to Seydel[6] metastatic growths in the lungs due to cancer occur approximately ten times as frequently as the primary growths. If the primary tumor is a sarcoma, the most common origin of the growth is the marrow of one of the long bones. Two tumors which histologically resemble both carcinoma and sarcoma are the *hypernephromas* and *malignant deciduomas;* both the hypernephromas and the deciduomas are especially apt to metastasize to the lungs. Of 22 cases of hypernephroma collected from the literature by Woolley[7] metastasis to the lungs occurred in 13. Malignant deciduoma involves the lungs secondarily in about 50 per cent. of the cases and in some instances first manifests itself by coughing, pleuritic pain, hemoptysis, and other symptoms referable to the chest.[8]

The Mediastinum.—Owing to the variety of the mediastinal contents and the close relationship important structures bear to each other in this confined space the occurrence of a tumor whether benign or malignant is a serious matter. Comparatively small benign tumors may produce most serious symptoms. Tumors comprising the benign group are individually quite rare and the aggregate number of all benign tumors is not large. Among the benign tumors encountered in the mediastinum may be mentioned the following: fibroma, lipoma, chondroma, myoma, simple cyst and dermoid cyst and teratoma. Of the benign tumors *dermoid cysts* are by far the most common. Hertzle[9] has reported 6 cases and collected 72 cases from the literature.

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[1] *Virchow's Archiv,* Band cxiv, S. 191, 1896.

[2] *Münchner Med. Woch.,* lvii, No. 9, 1910.

[3] *Brit. Med. Jour.,* Feb. 14, 1903.

[4] "Diseases of the Bronchi, Lungs and Pleura," 1915.

[5] *Virchow's Archiv,* cxxi, 156, 1913.

[6] *Loc. Cit.*

[7] *Amer. Jour. Med. Sc.,* cxxv, 1903.

[8] STEVENS, *Am. Jour. Med. Sc.,* cxliv, 1912.

[9] *Am. Jour. Med. Sc.,* August, 1916.

By far the commonest tumors involving the mediastinum are t comprising the malignant group. Hare in 1889 collected 520 insta of mediastinal affections and of this number he classed 134 as carcin and 98 as sarcoma. Many of the cases included in his analysis reported at a time when the histological distinction between the types of malignant tumors was not fully understood. Now that nature of these tumors is better known it is recognized that sarcon much more common than carcinoma. Endothelioma rarely invades mediastinum primarily; it may extend from a primary pleural growth

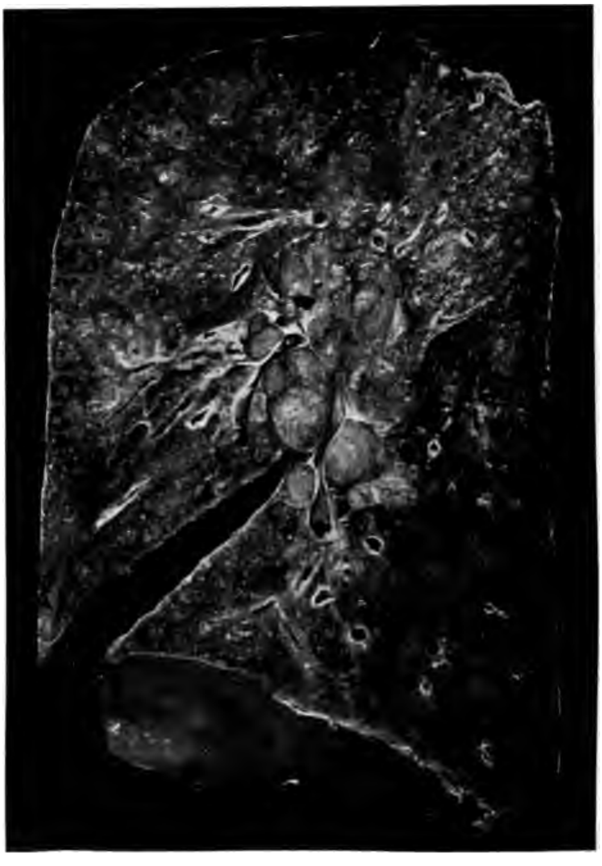

FIG. 320.—Colloid carcinoma.

Morbid Anatomy.—Benign tumors occurring in the lungs or bron are quite rare. They usually involve some one of the mediastinal stru tures. As has already been stated a tumor involving either the lungs bronchi is usually malignant. It is frequently asserted that in prima carcinoma of the lungs and bronchi that the right side is more freque involved than the left. Adler[1] states that the difference in the invol ment of the two sides is too small to serve as a basis for a theory. We

[1] "Primary Malignant Growths of the Lungs and Bronchi," 1911.
[2] *Loc. cit.*

hat in 69 cases the two sides were involved with approximately 'equency. Primary sarcoma is also quite as apt to occur on one the other.

ıary malignant growths may occur as a single large tumor having n in the root of the lung and extending into the pulmonary tissue : may be a number of small nodules varying in size from a hazel- a small orange. Less commonly the lung may be studded with

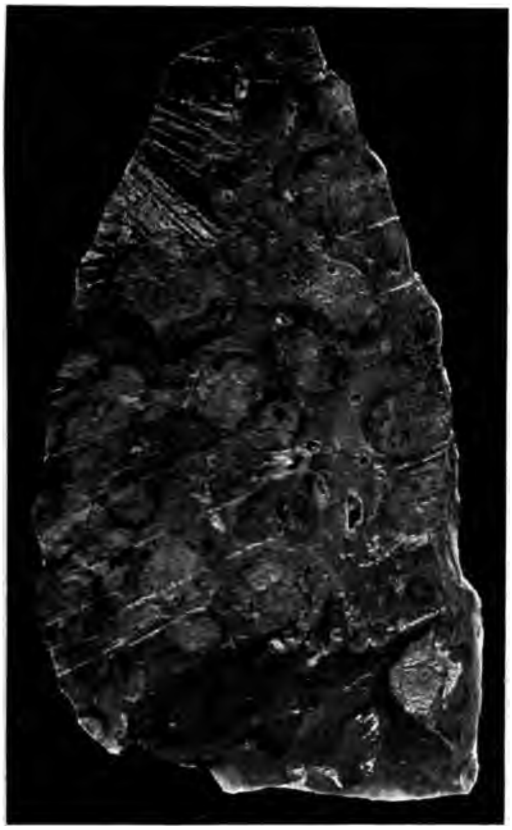

FIG. 321.—Sarcomatosis.

odules resembling miliary tubercles. This widespread distribu- metimes referred to as carcinomatosis or sarcomatosis, as the case , is seen more frequently as a result of metastasis (Figs. 320 and Occasionally the growths are almost entirely subpleural. Rarely ıor may be single and found projecting into the lumen of one of ger bronchi.

ead of appearing as an isolated tumor formation the growth may ı an infiltrating form. Wilson Fox describes two types of infiltra- First, it may occur as a general infiltration in which large areas ung are involved. This may be uniform or here and there traces ıonary tissue may remain intact. The process closely resembles a dense tuberculous infiltration (see Fig. 325). Secondly, the infiltration may radiate from the root of the lung by way of the lymphatic channels attending the bronchi. Small or large tumor nodules or masses may be seen surrounding the bronchi. In some instances the bronchi are but little involved and the lung tissue is chiefly affected.

The carcinomatous growths are of a white, grayish or grayish-yellow color and are of firm consistency. When they occur in the lung tissue they are apt to be softer, and may break down and empty into a bronchus thus forming a cavity. The most common form of cancer is that composed of cylindrical cells. Metastases to other organs in the body are quite common. Bronchial carcinoma is especially prone to give rise to metastasis to the brain (Weller).

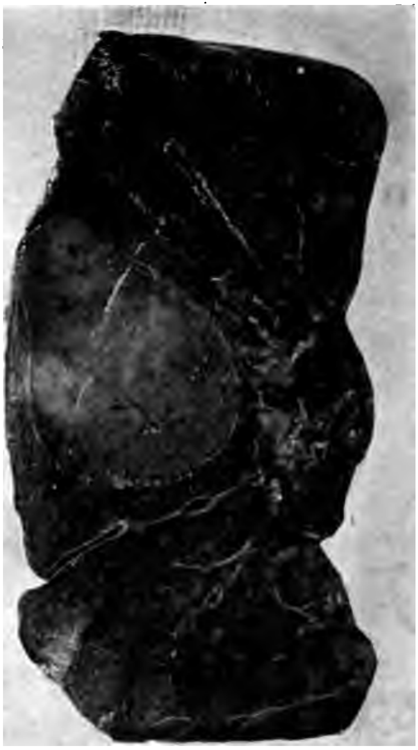

FIG. 322.—Large metastatic growth in right lung. Primary growth a hypernephroma of left kidney.

Sarcomatous growths usually occur in the form of isolated tumors which may be single or multiple. Large single growths sometimes attain the size of an infant's head (Fig. 322). Infiltrating growths are less common than with carcinoma. The tumors are usually somewhat soft and of a grayish, whitish or reddish color. Any type of sarcoma may be found but the small round-celled variety is the most common. Metastasis occurs but is less common than in the case of carcinoma. Other changes in the lungs may consist of congestion or aletectasis in the tissue immediately around the tumor masses. Less frequently bronchopneumonia, abscess or gangrene of the lung may arise. A pleural effusion is not uncommon especially when the pleura is involved. Effusions which arise as the result of malignant disease not only have a marked tendency to reaccumulate after removal but are also often hemorrhagic in character. The last feature is always suggestive of malignancy.

While there are some who believe the association of *tuberculosis* and malignant disease of the lungs is not infrequent the majority of observers hold the view that the two diseases rarely coexist. Among the 662 autopsies at the Phipps Institute there has been no instance in which the two diseases occurred together. K. Wolf[1] has reported 31 cases of malignant disease of the lungs in 13 of which tuberculosis also existed. Shaw[2] however, accepts but 8 of these as being even possible and regards most of them as doubtful. He furthermore holds that the only true test is the demonstration of tubercle bacilli. Among 60 cases of malignant disease studied by Ross[3] there were 2 cases in which tubercle bacilli were found in the sputum. He states that the frequency of healed tuberculous lesions found at the autopsy is of no significance as they are found with equal frequency in individuals dying of other diseases.

Secondary involvement of the lungs may occur as the result of extension from a nearby source or as the result of metastasis from a distant focus and while the lungs may escape it is rare for the metastatic growths to occur in the lungs alone. In the majority of cases the primary tumor is located in the breast, esophagus, stomach, liver, peritoneum, testes and bones. The secondary growths usually occur in the form of large or small isolated nodules. There may be only one or they may be multiple. Rarely the secondary invasion takes the form of a diffuse infiltration and still more rarely carcinoma may occur in a miliary form (carcinomatosis). As a rule, the histology of the secondary tumors is that of the parent growth.

Malignant disease involving the pleura may be primary or secondary as the result of direct extension from the lung or as part of a general metastasis. When primary the most common form is an endothelioma. When malignant disease affects the pleura primarily it manifests itself in two forms. First, it may consist of an infiltrating process which greatly increases the thickness of the pleura. Not uncommonly the parietal layer is involved exclusively without implication of the chest structures but extending into the mediastinal tissues (Wilson Fox). If limited to the pleura the change may be attributed to simple inflammatory thickening. Secondly, the growth may consist of large or small nodules in both layers of the pleura (Fig. 323). The infiltrating and nodular forms may occur together. Direct extension of the disease from the lungs or mediastinum may cause the same changes.

Mediastinal tumors of the benign types are of pathological interest only and during life are not to be distinguished one from the other with the exception of dermoid cysts.

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[1] *Fortschritte der Medicin*, 1895, xiii.

[2] *Brit. Med. Jour.*, 1901, i, 1831.

[3] *Edinburgh Med. Jour.*, December, 1916.

The *sarcomatous and carcinomatous growths*, on the other hand, present characteristics which render it possible to make a correct diagnosis of the malignant nature of the tumor in many instances. A sarcoma may spring from the connective tissue in the mediastinum, the peribronchial or the mediastinal lymph nodes. Tumors in this situation are usually of the spindle-celled type or lympho-sarcoma, the latter being the more common. The tumor usually consists of a solid mass which by its extension exerts more and more pressure on the mediastinal contents. It may extend beyond the limits of the mediastinum and invade the lungs secondarily. Tumors situated in the anterior mediastinum very often cause a bulging of the chest wall in the median line or to one side or the other of the sternum.

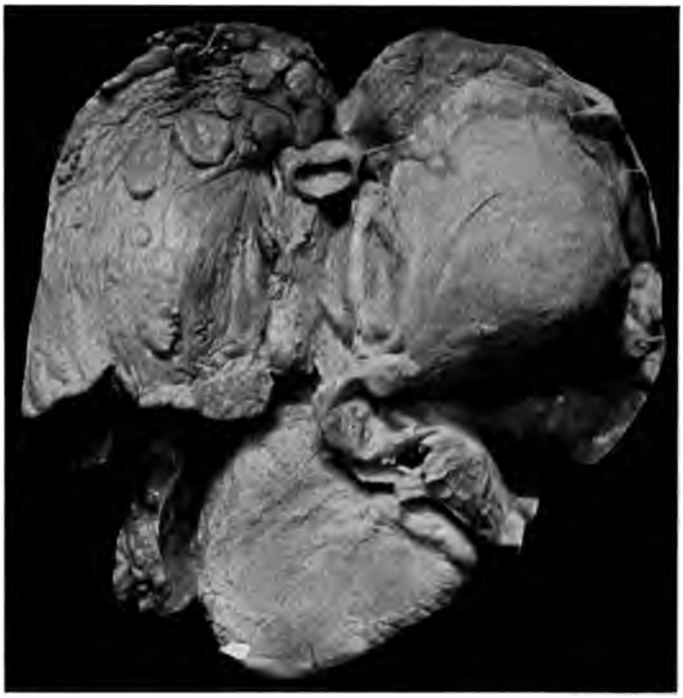

FIG. 323.—Sarcomatosis of pleura.

The consistency of these tumors is moderately firm and they are of a gray, yellowish-gray or reddish color. Rapidly growing tumors are apt to be quite soft and friable.

A primary carcinoma is not common in the mediastinum. If we eliminate cases which should be classed as sarcomas, lympho-sarcomas, secondary carcinomas, pleural endotheliomas, and bronchial carcinomas, few cases remain (Christian). A mediastinal growth occurring in middle aged people and which tends to spread to surrounding structures as indicated by the symptoms, physical signs and roentgenograms, is probably a carcinoma. The final proof, however, must rest on the histological examination.

ms.—From the clinical standpoint the benign tumors can be from consideration. With one exception they are of patho- erest only. Dermoid cysts will be considered separately.

oms of malignant growths may be divided into three classes: which occur in all forms; (2) constitutional symptoms; and symptoms which indicate either the nature or the location of

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ET.—The onset may be insidious with a gradual failure of it may be more or less acute with severe symptoms and all ces of a rapid decline.

a, cough, expectoration and pain are the most common

a is one of the earliest and one of the most common symptoms d in intrathoracic growths. It may be due to destruction of iary tissue by the tumor. On the other hand, it is to be borne iat intense dyspnea may occur out of all proportion to the damage demonstrable in the lungs. Cyanosis is commonly der these circumstances. In a case seen in the Phipps Insti- itient's only symptoms were intense dyspnea, cyanosis and a ;h. Examination of the lungs revealed a few scattered râles des, but no localizing signs. The heart showed nothing ab- l the pulse rate was under 90. At the autopsy not more than n nodules, the size of an English walnut, were found in each primary disease consisted of a small scirrhous carcinoma in ast. The cause of the dyspnea and cyanosis in such cases is tood. Dyspnea may occur as the result of pressure on the chi or trachea in the case of mediastinal tumors. In some is believed to be due to pressure on nerve trunks. It may be as the result of a large complicating pleural effusion.

fficulty in breathing is usually constant but it may be par- character. Occasionally it is severe enough to be termed

tion to the dyspnea the breath sounds may have a stridulous le result of pressure on the main bronchi or trachea. Hoarse- ccur also if the left recurrent laryngeal nerve is involved.

—This is an almost constant symptom; in Walshe's experience iably present when the lungs are involved. It may be due iated bronchitis or as the result of pressure. It varies greatly

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ration.—The sputum may be mucoid, mucopurulent, blood- r hemorrhagic. The presence of blood in the sputum or the of small hemoptyses is extremely common when the lungs ed. Occasionally the expectorated blood is thick, dark in closely resembles currant jelly. When present it is highly of malignant disease.

s of suspected malignant disease of the lungs and bronchi the pecially when hemorrhagic in character, should be examined oscopically and microscopically. As the result of necrosis and bits of the tumor mass may be discharged in the sputum and whitish, grayish or pinkish-colored particles or shreds. These y resemble small pieces of washed-out meat (Salter). In the ecimen there may be present a large number of cells, either isolated or in clusters, which are very suggestive of malignant disease. So too is the presence of refractive spherical bodies containing fatty granules (Lenhartz). In a case seen several years ago the presence of numerous clusters of large cells in the sputum gave the first clue as to the nature of the trouble.

Pain may be the first symptom. It is usually an indication that the pleura is involved either primarily or secondarily. It may arise, however, as the result of pressure. Tumors in the mediastinum may, by extending forward, give rise to pain similar to that occurring in thoracic aneurism. The pain may be constant or paroxysmal. In the pleural cases it may have all the features of an acute pleurisy while in the mediastinal cases it may produce a dull boring pain due to pressure and erosion. Neuralgic pain may also occur if the intercostal nerves are involved and associated with this there may be herpes zoster. In some instances the pain is objective entirely and can be elicited only by pressure with the palpating finger.

2. CONSTITUTIONAL SYMPTOMS.—Malignant disease of the thoracic organs give rise to the same constitutional symptoms as do malignant growths in any portion of the body. The progress of the disease may be exceedingly rapid or it may be relatively slow. Wilson Fox states that the duration of the disease ranges from four years to three weeks while Walshe found the maximum length of time to be twenty-seven months and the minimum three and a half months. The acuteness of the disease depends to a large extent on the rate of the growth and the amount of pressure it exerts. In the case of mediastinal growths the disease may have been present for some time before pressure symptoms finally direct attention to its presence. In practically all cases there is a more or less rapid loss in weight and strength and the patient becomes cachectic. Night sweats are common and an irregular type of fever is frequently present (Fig. 324). In common with other chronic wasting diseases, anemia, malaise, a capricious appetite and digestive disturbances are common.

Metastases to other portions of the body may take place and entirely mask the pulmonary lesion. Metastasis to the brain is not an uncommon occurrence in tumors originating in the bronchi. Death finally takes place as the result of asthenia, hemorrhage or an acute infection.

3. PRESSURE SYMPTOMS.—For the most part the special or localizing symptoms result from the pressure exerted by mediastinal growths. Pressure on one of the pneumogastric nerves may give rise to paroxysmal dyspnea or cough and occasionally to attacks of vomiting. The pulse may be increased in frequency or occasionally it may be greatly retarded. If the recurrent laryngeal nerves, especially the left, are involved by the growth there may be hoarseness or aphonia. Pressure on the sympathetic may lead to inequalities of the pupils, that on the affected side being contracted or dilated.

Symptoms referable to the blood-vessels are not unusual and, as a rule, the veins are more frequently affected than the arteries. In not a few cases inequality of the radial arteries has been noted. As the result of pressure on the superior vena cava or one of its main tributaries, the veins over the upper part of the chest may become greatly dilated, varicose and tortuous. In addition the face, upper part of the thorax and the arms may become cyanosed, swollen, and edematous. At times the of the neck and chest wall become brawny and very tense. The ces of venous stasis may be limited to one side or may involve sides. As the result of the interference with the returning blood

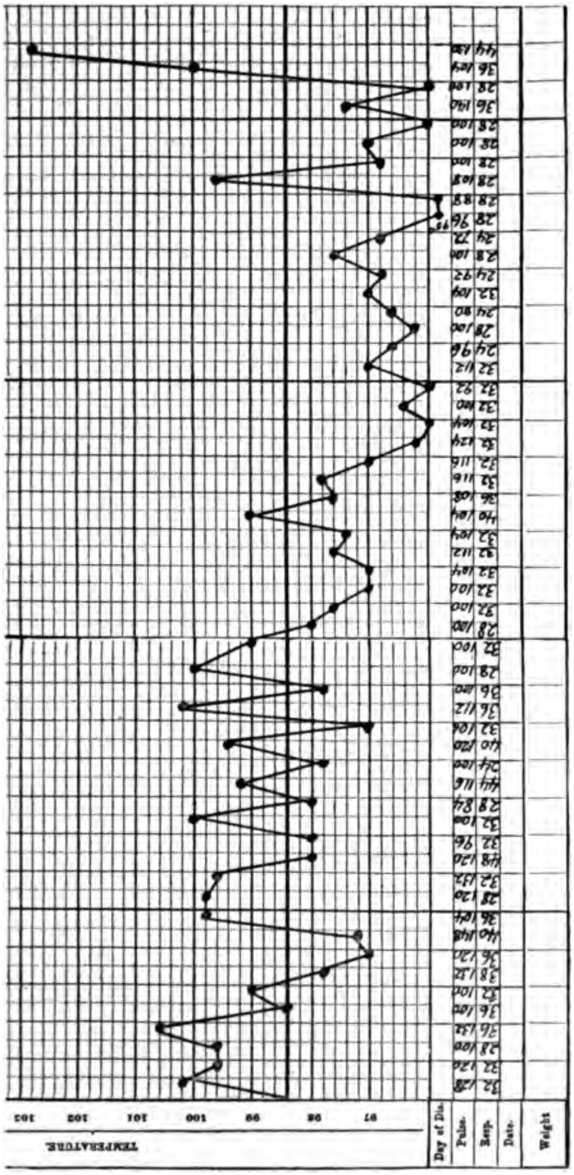

FIG. 324.—Carcinoma of the lung.

ral symptoms may occur, such as headache, vertigo and somce. These symptoms are apt to be increased by exertion and, in instances, are among the first manifestations of an intrathoracic h.

Pressure on the esophagus may give rise to difficulty in swallowing.

Dermoid Cyst.—As previously stated this growth is the only one of the benign tumors which is of clinical interest. The most common situation of tumors of this type is in the upper part of the chest behind the sternum. The cyst or tumor, as the case may be, may exist without giving rise to any manifestations whatever. When symptoms are present they are the same as those arising from any form of tumor, namely, cough, expectoration, pain and pressure symptoms. When it can be demonstrated that a tumor is the cause of the above symptoms a dermoid cyst or tumor is to be suspected if the progress of the disease is slow and long drawn out. This is in contrast to malignant tumors which are characterized, as a rule, by a rapid rate of growth. The most distinctive feature of dermoid cyst is the presence of hair in the sputum. In some instances the cyst ruptures through the chest wall in which case particles of hair may be detected in the discharge from the fistulous opening.

Secondary Growths.—The symptoms of secondary malignant growths are essentially the same as in the case of the primary growths. The diagnosis is oftener easier, however, because of the existence of a primary growth or the knowledge that a malignant tumor has been removed. Under these circumstances respiratory symptoms which might otherwise be misinterpreted are recognized as being due, in all probability, to secondary malignant growths.

Physical Signs.—Generally speaking the abnormal physical signs produced by intrathoracic tumors are elicited over the lower half of the chest except in the case of mediastinal growths which, although occurring most frequently in the upper half of the thorax, are situated behind or just to one side of the sternum. The apices are rarely involved except as part of a general invasion of the entire lung. In the metastatic cases physical signs pointing to pulmonary involvement are commonly absent or very indefinite. Physical signs indicative of a pleural effusion, on the other hand, are relatively frequent.

Inspection.—This may be entirely negative. In the case of mediastinal involvement pressure signs may be detected. The pupils may be unequal as the result of pressure on the sympathetic. Dilatation of the veins in the chest is common. Usually the veins over the upper part of the thorax are the ones involved but the veins of the arms, lower thorax and upper abdomen may be involved also. Venous stasis may also manifest itself by cyanosis of the lips and ears. Edema of the chest wall also occurs. If the growth is in the anterior mediastinum the upper part of the sternum may present a bulging point and this external tumor may pulsate. The pulsation is due to transmission of the aortic impulse. Localized bulging may be noted also in other portions of the chest, especially in rapidly growing tumors.

If the growth is unilateral the affected side will be seen to expand poorly. The apex beat of the heart may be displaced. In some instances extreme clubbing of the fingers with or without enlargement of the long bones (hypertrophic pulmonary osteo-arthopathy) may develop very rapidly (see Fig. 3). If the tumor originates in one of the large bronchi direct inspection of the growth is possible through the bronchoscope.

Palpation.—Over the involved area of the lungs the tactile fremitus is usually absent or greatly diminished. Palpation also serves to emphasize differences in the expansion of the two sides. Firm pressure with the finger tips will often elicit tender spots, especially if the pleura is involved. Pressure with the finger tips also may reveal the presence of edema of the chest wall. In some instances the superficially placed lymph nodes may show enlargement as the result of metastasis.

Percussion.—Areas of absolute dulness may occur at any point and are dependent on the nature and extent of the growth. Dulness is most commonly encountered directly over or to one side of the sternum, in the interscapular region and over the base of the lungs.

Auscultation.—If the growth is limited to the mediastinum there may be no noticeable auscultatory change. If pressure is exerted on the trachea or large bronchi the breath sounds may have a harsh and stridulous character. Involvement of the pulmonary tissue either primarily or secondarily may give rise to broncho-vesicular, bronchial or suppressed breath sounds. In the pulmonary cases râles are nearly always heard as the result of an associated bronchitis. With involvement of the pleura the breath and voice sounds are usually distant and suppressed.

X-ray Examination.—An X-ray examination is always desirable in cases of a suspected malignant growth. It has been shown by Carman and his co-workers at the Mayo Clinic that in the majority of instances the roentgen findings are distinctive and that a positive diagnosis is possible by this means when other methods only suggest malignancy. As has been pointed out by these observers, however, there should be a careful correlation of the roentgen findings with the clinical history and the physical and laboratory findings.

Diagnosis.—The recognition of primary malignant growths is not easy. This is due in part to their rarity and in part to the fact that the symptoms to which they give rise are encountered in the more common thoracic affections. Secondary malignant disease of the lungs on the other hand, is, in the majority of instances, easily recognized providing there is present a primary growth or the knowledge that such a growth has been removed. If, however, the primary growth escapes detection and gives rise to no symptoms metastasis to the lungs may occur without the true nature of the trouble being suspected. Pressure symptoms should always suggest an intrathoracic growth especially when aneurism can be excluded. The only distinctive feature of these growths is the presence of numerous large cells, singly or in clusters, in the sputum and this is available in the lung cases only.

Tuberculosis.—Instances of malignant disease are not infrequently seen in institutions for the care of tuberculous patients. Washburn[1] reports the finding of 5 cases of malignant disease of the lungs out of 198 autopsies at the Boston Consumptives Hospital. The fact that intrathoracic growths almost invariably give rise to symptoms which are associated constantly with tuberculosis is responsible for this. The persistent absence of tubercle bacilli in the sputum, the marked dyspnea and the localization of the physical signs in the anterior mediastinum, about the roots or at the bases of the lungs should serve as warnings. Tuberculosis is either localized at the summit of the lung or if the whole lung is diseased, the most destructive lesions are found where the process first started, namely, at the apex. Occasionally the malignant growth is confined to the upper lobe of the lung (Fig. 325). In such cases a diagnosis of tuberculosis can be avoided only by giving heed to the negative sputum examinations.

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[1] *Boston Med. and Surg. Jour.*, October 28, 1915.

Bronchitis.—Occasionally cases of malignant disease are encountered in which there are no localizing signs in the lungs and all that can be detected is the presence of scattered râles. If the sputum is blood-streaked and does not contain tubercle bacilli and in addition, the heart shows no evidence of failing compensation, malignant disease should be thought of. Extreme dyspnea and cyanosis occurring in a case presenting the physical signs of a simple bronchitis are very suggestive of malignant disease.

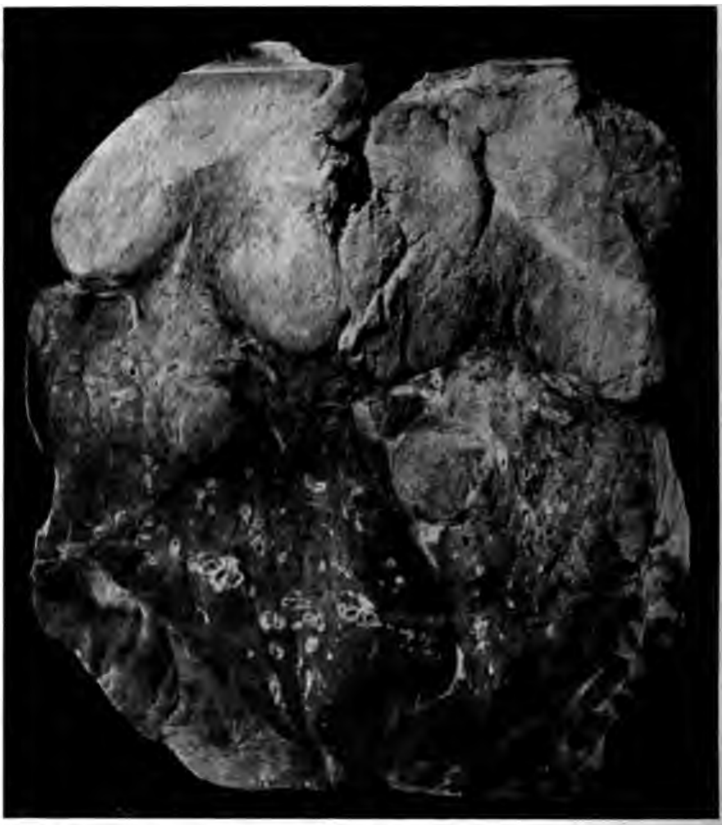
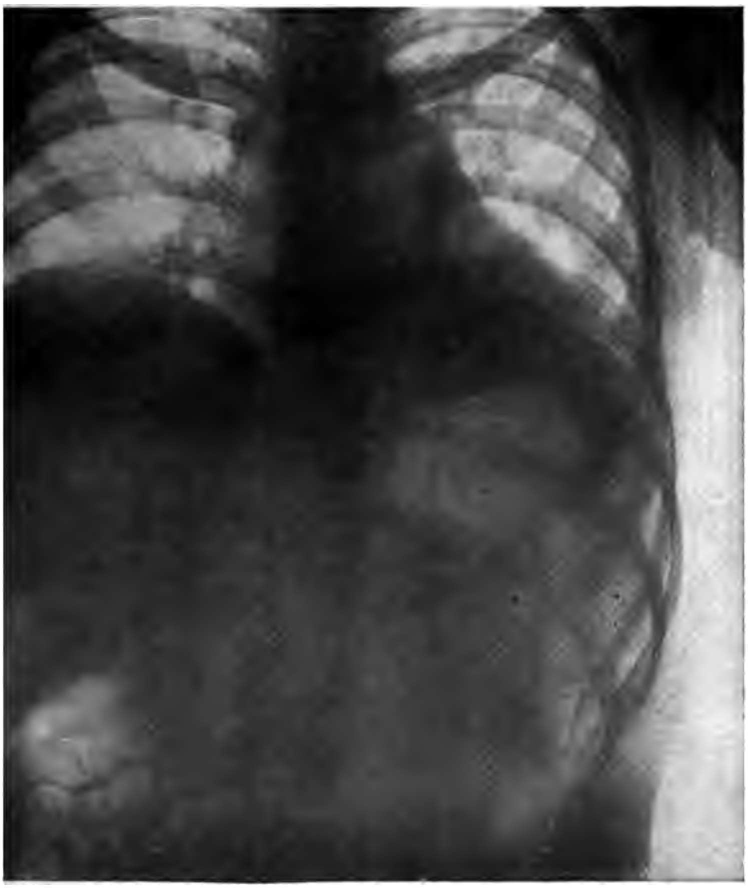


FIG. 325.—Malignant disease of upper lobe. Patient from whom this specimen came had been treated in several sanatoria for tuberculosis. (*Jefferson Medical College Museum.*)

Chronic inflammatory conditions of the lung are to be distinguished from malignant disease by their long duration and the absence of severe constitutional symptoms.

Pleural Effusion.—The frequency with which malignant disease is mistaken for a pleural effusion has long been recognized. The causes of the bulging of the affected side, the flat percussion note and e of the voice and breath sounds. Pleural effusion rarely o pressure symptoms although the displacement of the thoracic pecially the heart, is usually more marked than in cases of disease. The presence or absence of Grocco's triangle is of service in distinguishing the two conditions. The most thod is the use of an exploring needle. In addition the X-rays tilized in all doubtful cases.



umor in anterior mediastinum. Area of percussion dulness resembled that in pericardial effusion.

nant disease and pleural effusion coexist the exciting cause of ay be overlooked. A hemorrhagic effusion while not pathog- f malignant disease, is suggestive. Ross[1] in a study of 60 alignant disease involving the mediastinum stated that there sion in 29 and of this number 15 were hemorrhagic.

d effusions may be mistaken for malignant disease and *vice*

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[1] *Edinburgh Med Jour.*, December, 1914.

versa. The differentiation rests for the most part, on the use of the exploring needle, the X-ray examination and the course of the disease.

Pericardial Effusion.—A tumor involving the anterior mediastinum at times assumes the pyramidal shape of a pericardial effusion (Fig. 326). In case of an effusion the cardiac symptoms are usually marked while pressure symptoms are absent; the reverse is true in cases of tumor. As in all mediastinal affections the X-rays are invaluable.

Aneurism of the Arch of the Aorta.—Many of the symptoms and signs of aneurism also occur in tumors involving the anterior mediastinum. Thus the tumor may cause a bulging of the chest wall over the site in which aneurisms commonly occur and this bulging tumor may be seen to pulsate as the result of transmission of the normal aortic impulse through the tumor. Furthermore, the tumor may cause, by pressure on the main arterial branches, an inequality of the pulses. In the case of a tumor the pulsation is not expansile in character, a thrill is rarely present and a diastolic murmur is also very rare. In young individuals aneurism is much less common than tumor. An X-ray examination should always be made in doubtful cases.

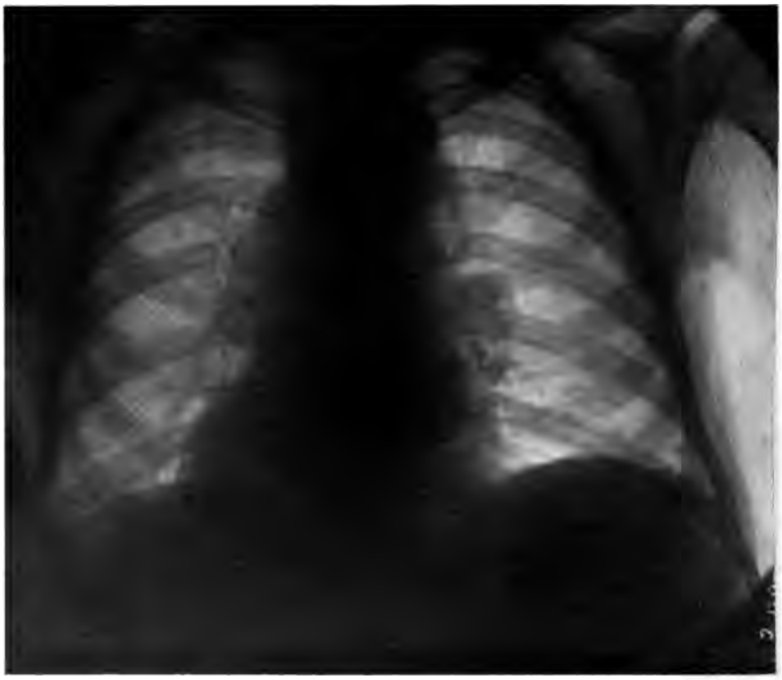

FIG. 327.—Mediastinal adenitis (in myelogenous leukemia). (*Dr. D. R. Bowen.*)

Leukemia and Hodgkin's Disease.—Involvement of the mediastinal lymph nodes often masks the picture of these conditions. The physical signs are those of a mediastinal tumor. The presence of enlarged lymph nodes elsewhere and the blood picture serve to differentiate them. The X-rays will also show the enlarged lymph nodes (Fig. 327).

INTRATHORACIC GOITRE

A not uncommon source of respiratory symptoms is an intrathoracic r substernal goitre. Since the introduction of the roentgen rays the resence of this type of goitre is easily detected. Formerly it is estimated hat not more than 50 per cent. were recognized prior to operation. C. I. Mayo[1] states that the majority of instances of ordinary or simple umors of the thyroid consist of two types—the colloid goitre or diffuse denoma and the encapsulated adenoma. The greater number of intrahoracic goitres are one of these two varieties.

The condition is always to be suspected in the presence of pressure ymptoms, as the size, appearance and density of the thyroid in the neck rarely sufficient to account for those symptoms.

There may be a feeling of suffocation on exertion or the patient may e subject to these attacks when lying down or when asleep. The feeling f suffocation is due to compression of the trachea. The voice may be usky or metallic in quality due to compression of a recurrent laryngeal erve. In addition to the dyspnea on exertion there may be swelling f the veins of the neck and cyanosis of the face.

On examination it is seen that the gland in the neck is usually that of moderate-sized tumor which seems to enlarge at its base or to disappear to the chest. As a rule it becomes pear-shaped with the larger end own. This is due to the fact that once the goitre gets into the chest, e funnel shape of the chest permits the lower end to expand, while e upper portion is contracted by the narrow thoracic aperture. As e intrathoracic portion enlarges, it tends to draw down the extrathoracic rt so that the latter may appear in the neck as a small nodule.

The veins of the neck and upper chest may be dilated and the breathg is that of an obstructive dyspnea. The upper lobe of the intrathoracic rtion may be felt above the clavicle. On percussion there is dulness er the upper portion of the sternum and this may extend to either side ward and toward the clavicle.

It is to be borne in mind that an intrathoracic goitre may be the cause pressure symptoms and give the signs of a tumor in the upper portion the chest without there being any enlargement of the gland in the neck.

ENLARGEMENT OF THE THYMUS

A number of terms have been employed to indicate an increase in e size of the thymus: Persistence of the thymus, hyperplasia, hyperophy, status lymphaticus, status thymico-lymphaticus, lymphatism, ymic tracheostenosis.

General Considerations.—Little is known concerning the thymus and ere is considerable difference of opinion as to its histological status. y some it is classed among the ductless glands, although structurally it s little resemblance to these organs; others look upon it as an epithelial ucture; the majority of observers, however, consider the thymus as longing to the lymphoid organs. *At birth* the thymus is seen to cont of a pinkish mass lying in the upper part of the anterior mediastinum d extending from just below the thyroid to the pericardium. It is mposed of two long, flat lobes more or less fused together, although often separated by a fissure. It is enclosed in a connective tissue capsule that sends in trabeculæ dividing it into lobes and lobules. The sac is loosely attached to the sternum in front, laterally to the pleuræ, and posteriorly is firmly united to the pericardium. Prolongations also connect it with the trachea, bronchi and pulmonary veins. At birth the thymus weighs from 7 to 10 grams. According to Warthin glands weighing 20 or 30 grams are to be looked upon as being abnormally enlarged. The thymus gradually increases in size up to the second year. It may then remain stationary or gradually undergo atrophy. With the advent of puberty it undergoes rapid involution and by the twentieth year is represented by a mass of fibrous and fatty tissue (see Figs. 65, 111, 113, 114).

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[1] Collected Papers of the Mayo Clinic 1910, p. 469.

The *function* of the organ is equally obscure. Krehl is of the opinion that its entire anatomical development, its persistence through adolescence, its regression as growth nears completion—all speak for its rôle in the building of the body. But although there has been considerable experimental work done in the endeavor to throw light upon the subject, nothing definite is known. It has been shown experimentally that there is, apparently, some relationship between the thymus and the testes, castration delaying the involution of the thymus while removal of the latter causes a more rapid development of the testes. It is also believed that the organ is concerned in the development of the osseous and central nervous systems and is in some way related to the blood-making organs.

Although the thymus may be the seat of disease such an occurrence is extremely rare. Clinically the thymus is of interest chiefly because of enlargement of the organ.

Etiology.—The condition is encountered for the most part in infants and young children but it may occur in adults. In some cases there is apparently a relationship between the hypertrophy of the gland and other disease processes, indicating possibly a compensatory action. In a few instances the enlargement may be associated with an acute infection. In many cases there is no adequate explanation. Infants who die suddenly after an attack of dyspnea or who are found dead in bed are often the subjects of enlarged thymus. Clinically it is important to bear in mind that *adenoids* and thymus hyperplasia are sometimes associated. It has long been known that enlargement of the thymus often complicates any kind of *goitre*. Crotti[1] has reported 6 cases (5 adults, 1 infant) in which this combination occurred. He urges that all goitre cases and especially those suffering from hyperthyroidism, should be examined for the presence of an enlarged thymus, as the sudden death which occasionally takes place in Graves' disease may be due to this cause. Matti[2] collected from the literature 133 cases of hyperthyroidism in which an autopsy had been held, and in 98 cases (74 per cent.) a hyperplastic thymus was found. Hector MacKenzie is of the opinion that the association of Graves' disease and enlargement of the thymus is too frequent to be accidental. Occasionally enlargement of the thymus occurs in leukemia.

Morbid Anatomy.—In addition to enlargement of the thymus there may be alterations in adjacent structures due to pressure and developmental changes. In the majority of cases of enlargement of the thy the pathological picture is that of "status lymphaticus." The children are large for their age and often have large heads. The body fat is usually increased. Anemia is more or less well marked and the facies present a pasty appearance. The lymphoid structures throughout the body are usually increased. The external and internal lymph nodes, tonsils and solitary follicles in the intestines are hyperplastic and adenoids are commonly present.

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[1] *Jour. Am. Med. Assoc.*, Jan. 11, 1913; *Ibid.*, Feb. 22, 1913.

[2] *Deut. Zeit. f. Chir.*, 1912, cxii.

The pressure of an enlarged thymus is exerted, for the most part, on the trachea which in extreme cases may be almost completely stenosed. Pressure may also be exerted on the heart and to this may be ascribed some of the cases of sudden death. When respiratory symptoms are present they are always due to pressure on the trachea which is often severe enough to cause death by suffocation. In those cases in which there is no pressure on the trachea death probably is due to toxemia resulting from an overproduction of the internal secretion of the thymus and lymph glands (Osler).

Symptoms.—In not a few cases there are no premonitory symptoms, or if they have been present they have been overlooked. Sudden death from enlargement of the thymus is not an uncommon event in young children who have given no previous evidence of ill health. In children who are believed to have been "overlaid" by the mother the true cause of death is to be found, in some instances, in enlargement of the thymus.

Occasionally a new-born child is extremely cyanotic and the respirations labored and noisy. It may be revived but dies in a few hours. In such cases a greatly enlarged thymus is found to be present.

Sudden death, the underlying cause of which is an enlarged thymus, may also follow a severe fright, great emotion, a surgical operation or anesthetization. Warthin believes that a large proportion of deaths occurring in surgical anesthesia are due to enlargement of the thymus. Suspected cases should be carefully examined before submitting them to an operation.

The most striking and important symptom of hyperplasia of the thymus is dyspnea due, in part, to diminution of space in the upper thorax and, in part, to varying degrees of stenosis of the trachea. The severity of the dyspnea varies tremendously. It may be nothing more than a mild form of stridor or it may be fulminating in character and cause death in a few minutes. Between these extremes all possible grades may occur. The difficulty in breathing often manifests itself in the first weeks or months of life and diminishes after the second year at which time the gland begins to undergo involution.

The dyspnea may consist of a stridorous type of breathing which may be audible at a distance. It is most marked during inspiration but may occur during expiration also. This type of breathing may remain stationary or it may become progressively worse as the pressure on the trachea becomes greater.

In more severe cases the disturbance in breathing is often paroxysmal
thmatic in character. The pseudoasthmatic attacks may occur in
with persistent stridor or they may develop when there have been
evious symptoms. This type of breathing was first described by
, and is often referred to as *Kopp's asthma*. A fatal issue may
v the first of these attacks but as many as twenty or thirty may
. "In paroxysmal dyspnea, the child suddenly suffocates and

becomes cyanotic. The stridor is extremely marked, respiration becomes accelerated and there is a sucking in of the suprasternal and infrasternal fossæ. The choking spell may occur without cause, or when the child cries from pain or anger. Hyperextension of the head or dorsal decubitus exaggerates the dyspnea. This would explain why dyspnea is more marked during sleep than during waking hours" (Crotti). Finally, there is the fulminant type in which death occurs suddenly without a history of previous respiratory trouble.

In the absence of respiratory symptoms hyperplasia of the thymus should be suspected in children in whom the body fat is in excess, who are anemic, thick-skinned and who have facies of the adenoid type. The superficial lymph nodes will often be found to be unduly enlarged, the spleen is palpable and the tonsillar tissue abundant. A blood count will frequently show an excess of lymphocytes.

Physical Signs.—*Inspection.*—The general appearance has been referred to. In many, if not the majority, of children suffering from an enlarged thymus there are evidences of rickets. Occasionally there may be some bulging of the upper part of the sternum and in extreme cases a tumor may be seen in the suprasternal notch during forced expiration.

Direct inspection of the upper air tract will reveal, in many cases, hypertrophy of the tonsils and the presence of adenoids; occasionally the stenosis of the trachea can be seen.

Palpation.—The gland may be felt in the suprasternal notch. Enlargement of the superficial lymph nodes and the spleen may be present.

Percussion.—This gives the most important information. The area of dulness is triangular in shape and can easily be determined by median percussion over the manubrium. The dulness extends beyond the edges of the sternum and is nearly always more marked to the left than the right. On the left side the dulness can be elicited in the first and second interspaces and occasionally in the third; on the right in the first interspace and less frequently in the second. The dulness may merge into the heart dulness below (Fig. 328; see also Figs. 111, 112, 113 and 114).

Jacobi[1] and Boggs[2] claim that the thymus is movable and that the mobility of the organ is a valuable diagnostic sign. Jacobi believes that in the dorsal decubitus the organ falls away from the chest wall. He, therefore, percusses the area over the thymus when the patient is lying on the back. On reversing the decubitus and percussing from underneath, the area of dulness will be increased if the thymus is enlarged as the gland has fallen back against the anterior chest wall. Boggs, from clinical observations, has determined that the gland moves up and down. It can be made to shift upward by hyperextension of the head in the middle line. He examines the patient in the sitting posture and determines the lower limit of dulness, then by extending the head the dulness will be found to have shifted upward, often as much as an interspace or more. Park and Maguire[3] criticize both these hypotheses on the ground that the capsule is so firmly attached to surrounding structure as to preclude movement. They suggest, however, that the thymus may be displaced inside its own capsule.

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[1] "Therapeutics of Infancy and Childhood," 1908.

[2] *Trans. Assoc. Am. Phys.*, 1911.

[3] *Arch. Int. Med.*, September, 1912.

While some assert that the X-rays fail to confirm the percussion ea of dulness, others have found that the two methods agreed. Hochnger found enlargement of the thymus by both percussion and radioraphic methods in 26 out of 58 children examined. Crotti insists that e percussion findings should always be checked up by the X-rays. If nlargement of the thymus is present, a more or less triangular shadow with sharply defined edges is seen in the superior mediastinal space. It overlaps the normal mediastinal shadow and extends upward from the base of the heart. In some cases, it is superimposed on the base of the heart like a cap.

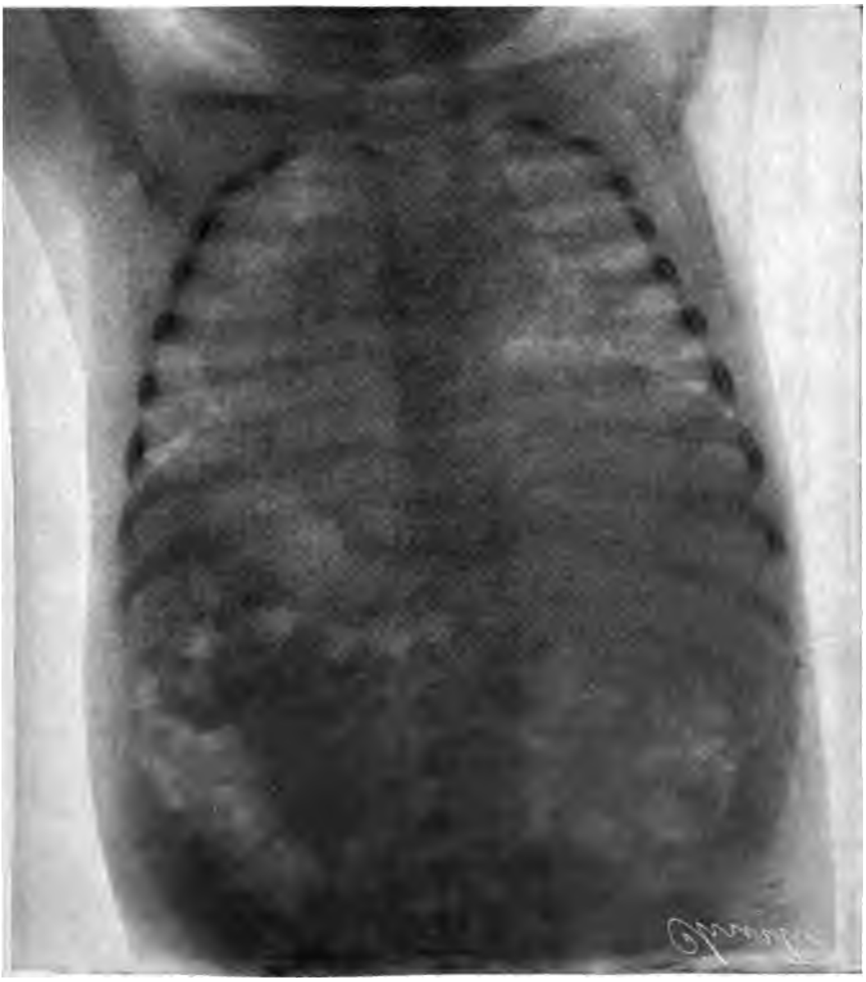

FIG. 328.—Enlarged thymus. (*Roentgenogram by Dr. H. K. Pancoast.*)

Auscultation.—If respiratory disturbances are present they can usually be detected without the aid of the stethoscope as the stridor may be audible at some distance from the body. While heard all over the chest the stridor is more intense over the upper part.

Diagnosis.—In children the most important symptom is alteration in the character of the breathing. This consists of a persistent stridor both inspiratory and expiratory but most marked on inspiration. Children who are subject to this type of breathing may suffer from paroxysmal suffocative attacks resembling asthma. In some instances these so-called asthmatic attacks occur at intervals, the breathing being normal at other times.

Congenital stridor may be due also to malformation of the vestibule of the larynx. In this condition the difficulty in breathing is entirely inspiratory. The condition may be recognized by direct inspection with the laryngoscope or by digital examination.

Enlarged tuberculous lymph nodes about the root of the lung may give rise to stridor. This type of breathing when due to enlarged lymph nodes rarely appears before the fourth month. In tuberculous adenitis the radiograph gives an entirely different picture. The trouble is seen to be at the roots of the lungs, the enlarged lymph nodes showing as distinct isolated shadows.

In adults suffering with *goitre*, especially the exophthalmic type, it is important to have in mind that a persistent thymus may be present. The differentiation between an enlarged thymus and an intrathoracic goitre may present some difficulty. In goitre there is an up and down movement synchronous with swallowing which may be detected by the fluoroscope. This up and down movement is characteristic of goitre and occurs in no other condition.

HERNIA OF THE LUNGS (Pneumonocele)

This is a rare condition: not more than one hundred cases have been recorded in the literature. Three types of cases are recognized: congenital, traumatic and spontaneous.

Congenital hernia is the result of some developmental defect in the chest wall. As the lungs are in a state of collapse during uterine life there is no evidence of the trouble until some weeks or months after birth. Occasionally the condition does not manifest itself until adult life. A congenital hernia is most apt to appear in the lower intercostal spaces near the junction of the cartilage with the rib.

Traumatic pneumonocele is exceedingly rare, compared to the great number of penetrating wounds of the chest. When an open wound of the chest occurs it is followed, almost invariably, by collapse of the lung and the formation of a pneumothorax. Occasionally, however, and under circumstances which are difficult to explain, a portion of the lung protrudes through the opening. Makins[1] in an extensive experience with chest wounds, observed but one case in which this accident had occurred; and during the Russian-Japanese War only five cases were reported among 20,000 wounds of the chest (Adams). The condition is recognizable at a glance. In addition to open wounds the chest wall may be weakened by a severe strain, a blow, or a crushing accident. Although operations on the chest wall, at times, leave a point of weakened resistance through which the lung tissue may bulge, it is noteworthy how seldom this accident occurs. The hernia may develop very quickly or considerable interval may intervene (consecutive hernia).

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[1] "Surgical Experiences in South Africa, 1899–1900."

Spontaneous or non-traumatic pneumocele results when a weakened portion of the chest wall gives away under the strain of increased intrathoracic pressure. This may be produced in a variety of ways, such as ong-continued coughing, which is the most common cause, lifting, blowing a wind instrument, crying, etc. It usually occurs in the anterior or lateral aspect of the chest and only rarely posteriorly.

The hernia varies greatly in size ranging from one the size of an almond o a clinched fist and with few exceptions is unilateral. The hernia may e enclosed in a sac formed by the pleura. It can often be replaced and hen this is the case the opening in the chest wall may be made out readily. s a rule there are no symptoms, but in some instances, during the initial eriod of development, there may be pain. This may be aggravated by eep breathing or coughing.

The tumor consists of a rounded mass which is soft, elastic and crepiant. It may be covered with smooth, normal skin or, as in the case of a ost-operative hernia, by scar tissue. Coughing, deep breathing or change f posture may cause the tumor to increase in size. Over the tumor the ercussion note is resonant or tympanitic, the breath sounds vesicular nd the vocal and tactile fremitus are increased. In some instances the ernia may appear only intermittently. Strangulation, although rare, as been reported.

The following conditions may be confounded with a pneumonocele: ubcutaneous emphysema, empyema necessitatis, aneurism or tumor. Supraclavicular bulgings, which are not infrequently seen in patients vith emphysema and in emaciated tuberculous patients with distressing coughs of long duration, are not, as a rule, instances of true hernia of the lung, although a true hernia may occur in this situation.

Subcutaneous emphysema may cause a localized swelling on the chest wall, which on palpation crepitates and on percussion gives a tympanitic note. The crepitation, which is caused by the movement of bubbles of air in the tissues, may not only be felt but may be audible as well. The condition may be due to some gas-producing organism, such as the gas bacillus of Welch. More frequently it follows a punctured wound, traumatic or operative. It is not uncommon, for instance, after paracentesis. Subcutaneous emphysema is often seen in the lower part of the neck and over the manubrium as the result of trauma or a violent coughing paroxysm. A number of instances of this condition has been reported in association with influenza pneumonia. It appears first in the neck and may spread downward over the chest and abdomen.

In *empyema necessitatis* the external tumor may increase or diminish in size during coughing or forced breathing. The differentiation, as in he case of other tumors of the chest wall, depends on the presence or bsence of crepitation and the other signs commonly found in hernia. have seen one case of tuberculosis in which ulceration had destroyed a ortion of the intercostal muscles, leaving nothing but the skin and ubcutaneous tissues. At this point there was a distinct bulging on spiration and especially after coughing. It had every appearance of hernia but lacked the characteristic crepitation.

ALTERATIONS IN THE EXTREMITIES DUE TO CHRONIC PULMONARY DISEASE

A curious feature of chronic pulmonary affections is the frequency with which deformity of the fingers, toes and nails occurs. These changes are seen also in congenital heart disease; occasionally there is no demonstrable factor.

THE NAILS

In pulmonary tuberculosis abnormalities involving the nails are extremely common. The abnormality to be noted may consist of a change in the color, in the contour or in the texture of the nail. In cases of advanced phthisis, the nails are often livid and may be slightly cyanotic in appearance. In the early stages of tuberculosis the nails may be of an exaggerated pinkish color, closely resembling those which have been stained with a manicuring paste, or they may have a slight violet tinge. In either case these changes are to be looked upon as an evidence of serious infection. I have repeatedly seen these changes in the nails of individuals in whom the disease steadily progressed from bad to worse, although when first seen the amount of pulmonary involvement spoke for a favorable prognosis.

An extremely common change, especially in cases of tuberculosis, is curving of the nails. This is usually an exaggeration of the normal curve from side to side so that the nail appears narrower than normal. Not infrequently the lateral curving is associated with a turning downward of the distal portion of the nail, over the end of the finger. When clubbing of the fingers also is present the curving downward of the nail over the end of the finger is usually very marked. Among 3551 patients observed at the Phipps Institute curving of the nails was noted in 1382 or 38.9 per cent.

Longitudinal and transverse striations are quite common. The transverse striations often correspond with exacerbations of the disease and are similar to those occasionally seen after an acute illness of any kind.

An unusual change, occasionally noted in those suffering from some chronic pulmonary affection is the so-called "oyster-shell" nail. Hirshberg[1] first called attention to this change. Five cases have been seen at the Phipps Institute. The nail becomes loosened from its bed laterally and distally, becomes very brittle and is easily broken. It is, also, deformed by ridges and crater-like depressions, giving it the appearance of an oyster shell. Both the fingers and toes are involved.

CLUBBING OF THE FINGERS AND TOES

This curious deformity of the fingers was known to the ancients was in later times referred to as the "Hippocratic deformity of fingers." The toes are often involved but never to the same extent the fingers. Occasionally the nose assumes a bulbous or clubb appearance.

Clubbing of the fingers is very common in cases of pulmonary tub culosis. It is seen in its most exaggerated form, however, in bronchi tasis, empyema and congenital heart disease. In the ordinary chro f pulmonary tuberculosis, the clubbing is never extreme; when it is n be reasonably certain that the tuberculosis is associated with d fibrosis of the lung and dilatation of the bronchi. In a group of s seen at the White Haven Sanatorium extreme clubbing of the s was noted in 10 of the 21 reported.[1] In these cases the primary e was a pulmonary fibrosis associated with dilatation of the bronchi, ing from the inhalation of inorganic dust. Later a tuberculous ion was superadded. Among 3551 cases of tuberculosis seen at the s Institute varying degrees of clubbing of the fingers was noted in 21.7 per cent. In extreme examples of the condition the associa- ith bronchiectasis is so frequent as to be of considerable diagnostic and the same is true of the association of marked clubbing and sis with congenital heart disease (see Fig. 1).

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[1] *Am. Jour. Dermatology*, August, 1911.

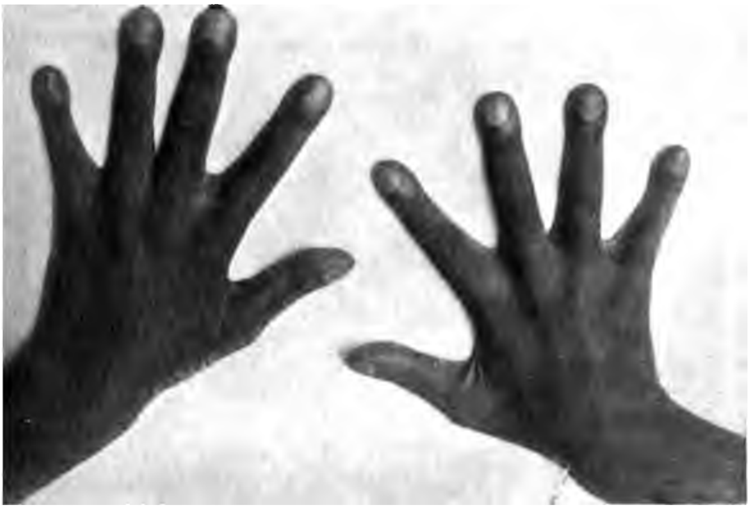

FIG. 329.—Simple clubbing.

the vast majority of cases the development of the clubbing is us and slow and usually passes unnoticed by the patient. Rarely elops quickly. West[2] records two cases in which the clubbing ped in two and four weeks respectively and Godlee[3] mentions a case ch well-marked clubbing appeared in two months. Aside from the nity there are no symptoms although occasionally pain is com- d of in rapidly developing cases. Once established the deformity manent although some improvement has been noted after the ge of an empyema.

simple clubbing the enlargement is, for the most part, due to a plasia of the soft parts, principally fat. Two clubbed fingers (shown . 329) which I had removed at autopsy and placed in Kaiserling's oth lost the deformity; Fowler had a similar experience with a d finger preserved in alcohol. Since the introduction of the

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[1] *Am. Jour. Public Health*, 1914, vol. iv, No. 9.
[2] *Trans. Clin. Soc.*, vol. xxx, 1897.
[3] FOWLER and GODLEE: "Diseases of the Lungs," 1898.

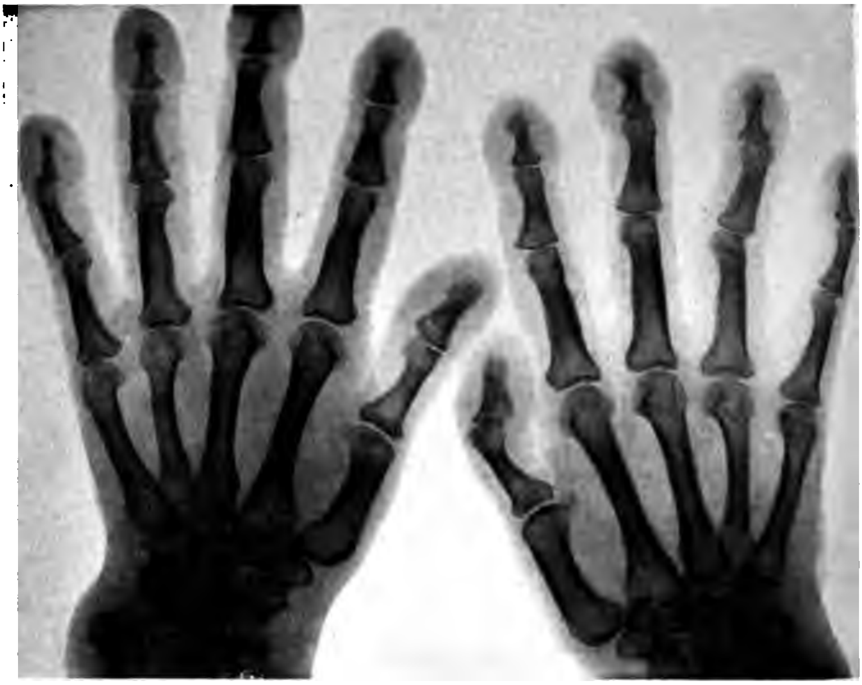

FIG. 330.—Radiograph of clubbed fingers in hypertrophic pulmonary osteo-arthropathy. "Burring" of terminal phalanges.

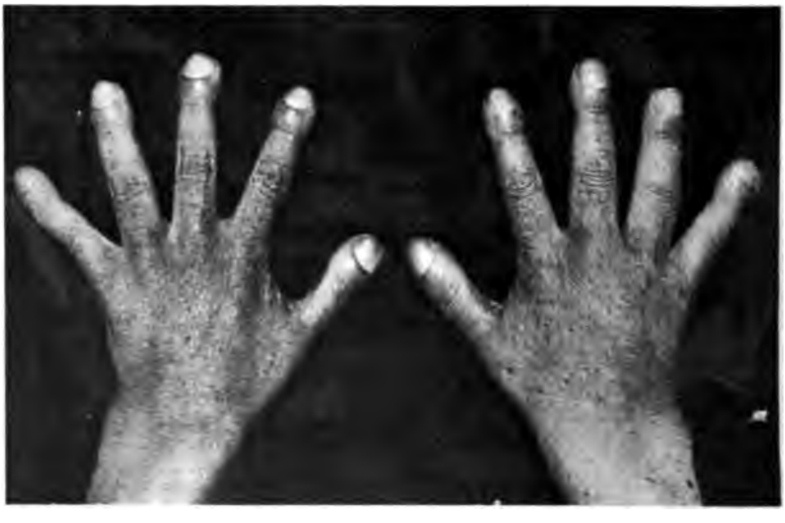

FIG. 331.—Clubbing in hypertrophic pulmonary osteo-arthropathy.

X-rays it has been shown that in many cases there is also some increase in he bony parts. Usually the change is limited to the terminal phalanx ut there may be present also some periosteal proliferation of the long ones. Locke[1] states that the Roentgen-ray examination of 39 cases f simple clubbing showed alterations of varying degree in the ungual halanges in 5 cases and periosteal proliferation of the long bones in 12. he change noted in the terminal phalanges consists of a proliferation f the distal half, usually giving a distinct chestnut-burr appearance Fig. 330), and occasionally immense burr-like processes are seen.

The affection is almost always bilateral and symmetrical and is said o take place first in the thumb and forefinger; later the other fingers also ecome clubbed. A few cases have been reported in which the deformity as unilateral and associated with an aneurism of the subclavian artery. uch cases are not instances of true clubbing but are to be regarded as a orm of edema due to interference with the circulation. The deformity onsists of enlargement and thickening of the digital pulp of the ungual halanx associated with curving downward of the end of the nail. This ype of nail is sometimes referred to as the "parrot-beak" nail. Owing to he bulbous appearance of the terminal phalanges, fingers so deformed ave been likened to the end of a club or a drumstick; Trousseau comared the enlargement to the head of a serpent (Fig. 331). The skin ver the bulbous portion of the finger is usually shiny and stretched in ppearance. In the rapidly developing type the swelling of the soft arts is extreme, the nail bed is deeply injected and swollen and the nails re red. This is especially apt to occur in cases of empyema; the most arked example which has come to my notice was in a case of recurrent arcoma with extensive metastasis to the lungs (see Fig. 3). In the lowly developing type the color of the skin and nail may be of a deeper ink than normal or it may be dusky and livid. When the clubbing is ssociated with congenital heart disease, both the nails and fingers are arkedly cyanotic (see Fig. 1).

"When true clubbing of the fingers is present, if the observer places ne of his fingers upon the upper edge of the nail at its free margin, *i.e.*, he distal end, and his thumb upon the under surface of the clubbed finger, nd presses downward, the hard margin of the root of the nail can be istinctly felt if another finger is applied over it" (Fowler).

HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

Attention was first called to this remarkable deformity by Bamberger[2] nd Marie.[3] The name of the latter has been especially associated with the ondition. Since the appearance of these original papers numerous studies f collected cases have been made. Among these may be mentioned the apers by Walters,[4] Thayer,[5] Janeway,[6] Wynn,[7] Ebstein,[8] and Alexander.[9] he most recent and the most complete study is that by Locke.[10]

[1] *Arch. Int. Med.*, May, 1915, Part I

[2] *Wien. Klin. Woch.*, 1889, ii, 225; *Zeit. f. Klin. Med.*, 1891, viii, 193.

[3] *Revue de med.* 1890, x, 1.

[4] St. Thomas Hospital Report, 1895.

[5] *Phila. Med. Jour.*, 1898.

[6] *Am. Jour. Med. Sc.*, January, 1903.

[7] *Birmingham Med. Review*, 1904.

[8] *Deut. Arch. f. Klin. Med.* [Festschrift], 1906, lxxxix, 68.

[9] St. Bartholomew Hospital Report, xlii, 1906.

[10] *Arch. Int. Med.*, May, 1915, Part I.

The original name has been subject to various changes and modifications. The following terms have been suggested: Secondary hypertrophic osteo-arthropathy, hyperplastic osteo-arthropathy, secondary hyperplastic ostitis, toxigenic ossifying osteo-periostitis, tuberculous polyarthritis. Next to the original name, secondary hypertrophic osteo-arthropathy has been most used. The only advantage it possesses is, that it gets over the difficulty that, in some cases, no pulmonary lesion has been found; on the other hand, it fails to cover those cases in which the disease is apparently primary. It seems to me that Marie's original name had best be retained as the others are just as cumbersome, and do not express any more clearly the conditions present.

Etiology.—Males are affected more frequently than females, the proportion being nearly 8 to 1. With two or three exceptions all the cases reported have occurred in the white race. No age period is exempt but the vast majority of cases occur in the third and fourth decades. The greater liability to pulmonary affections during the third and fourth decades doubtless influences the osteo-arthropathic change rather than the condition of the bones.

The condition is almost invariably secondary to some obvious primary disease, usually of the lungs or bronchi. Locke has been able to find but 5 cases which offer any definite positive evidences of the existence of primary origin. From a study of the reported cases Locke found 139 which meet all the requirements; to this number he adds 5 personal observations; making 144 in all. I believe that this does not by any means represent the true proportion. Doubtless many cases have been recognized and not reported, and probably a still larger number have gone unrecognized or have been looked upon as cases of ordinary clubbing of the fingers. Within the past three years, I have seen three typical cases.

The rôle played by chronic respiratory affections is well recognized. Of the 144 cases reported by Locke 113 were associated with disease of the respiratory tract; 6 with disease of the circulatory tract; 13 with disease of the alimentary tract, of which 6 occurred with hypertrophic biliary cirrhosis; 7 were associated with miscellaneous conditions and 5 were apparently primary. Of the individual diseases pulmonary tuberculosis heads the list with 30 or 20 per cent.; bronchiectasis 28 or 19 per cent.; malignant disease 10 or 7 per cent. and empyema 8 or 5 per cent. Although bronchiectasis stands second I am inclined to believe it is by far the most important disease as many of the cases of chronic tuberculosis are associated with marked fibrosis and dilatation of the bronchi. Why these various conditions should be associated with osteo-arthropathic changes is not at all clear. Various hypotheses have been advanced, the most probable of which seems to be that the changes are in some way dependent on toxic absorption; this was Marie's idea and also Bamberger's. That it is the most reasonable explanation is to be inferred from the fact that extreme clubbing of the fingers is almost exclusively associated with bronchiectatic cavities in the lower lobes. In this situation the drainage is poor and the secretions stagnate. When the dilated bronchi are confined to the apex of the lung clubbing of the fingers is rarely present as the drainage is good.

Morbid Anatomy.—The changes consist primarily in a slowly progressive, ossifying periostitis beginning usually in the distal ends of the diaphyses of the bones of the forearm and lower legs, later involving also the other bones of the limbs and even in some cases nearly all the remaining bones of the skeleton. The periosteum is thickened, more vascular than normal, and shows subperiosteal deposit of new bone. At first the new bone is sharply differentiated from the old shaft but at a later period the two become indistinguishable. The cortex of the bones shows sclerosis and thickening with diminution of the medullary cavity.

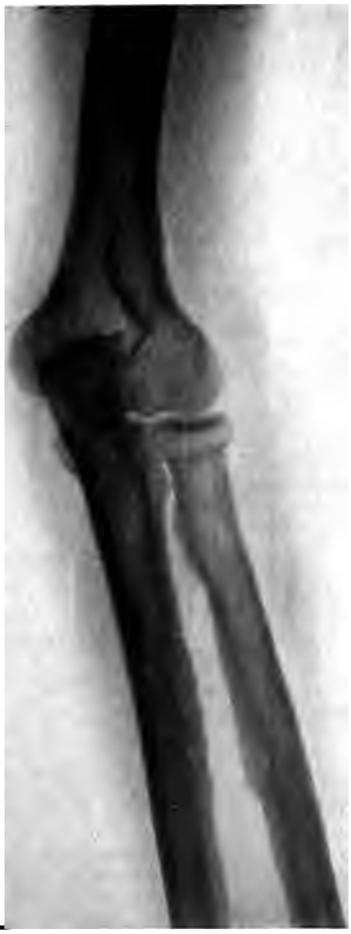

Fig. 332.—Radiograph of elbow-joint showing spicula of bone at upper end of ulna. Shafts of radius and ulna also show a roughened and uneven appearance similar to chronic periostitis.

The medulla is embryonic in character, with a tendency to fatty degeneration in its central part. Locke states that in the most extreme cases there remains scarcely any of the appearances of the original bone but instead the osseous tissue appears very thin, and of a coarse, irregular structure. In general such cases resemble osteitis deformans but without the curving or deformity, except in outline, which is characteristic of the latter disease. In addition to the subperiosteal new bone osteophytic growths are often present (Fig. 332). Hall[1] has reported a case in which affected bones were characterized by softness of texture, the mortem knife dividing them easily. The evolution of the bony cha can easily be followed by means of frequent examinations with the R gen rays.

In addition to the changes in the bones the joints show erosio the articular cartilages, periarticular thickening, and thickening of synovial membrane with an excessive amount of fluid. The soft p are also increased in size principally as the result of a hyperplasia of fatty tissue. Associated pathological changes have been descr in individual cases but none of them are constant and must be lo upon either as coincidences, or in some way related to the primary dise

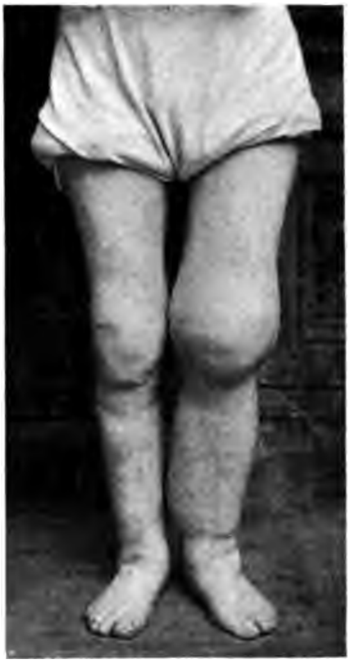

Fig. 333.—Enlargement of both legs in a case of hypertrophic pulmonary osteo-arthrop with large effusion in left knee-joint.

Symptoms.—The symptoms of hypertrophic pulmonary os arthropathy are almost entirely objective. In rapidly developing c pain in the hands is sometimes complained of and there may be pre also pain and stiffness in the affected joints. These symptoms may o during an acute exacerbation of a chronic case. In the great majo of cases the disease develops slowly and the patient is often unconsc of the changes until attention has been drawn to them. Briefly stated changes consist in a symmetrical enlargement of the hands, most ma in the finger tips, which are enlarged and bulbous, the so-called drums fingers. The nails are sharply curved downward, and resemble a par beak. Enlargement of the lower part of the forearm accompanies change in the hands. The enlargement is due partly to the formati new bone and partly to hypertrophy of the soft tissues. Analogous changes take place in the feet and legs. Locke has emphasized the fact that X-ray studies have shown that while the bones of the forearms and legs are most frequently involved the bones in any part of the body may be enlarged. Effusions into the joints are common, the joints most frequently affected being those nearest the bony changes, as the ankle, knee, wrist or elbow. Rarely the hip and shoulder joints are involved. The symmetrical distribution of the lesion is characteristic but occasionally one side is more involved than the other. This occurred in a case I[1] reported some years ago in which the changes in the hands and forearms were symmetrical but in the lower extremities one side was much larger than the other (Fig. 333).

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[1] *Edinburgh Med. Jour.*, New Series, vol. xviii, 1905.

Pain is rarely complained of but the greatly increased size of the hands may interfere with the finer movements, such as needle work or writing. Sweating of the palms of the hands and soles of the feet is common. Involvement of the joints may produce stiffness, pain or disturbance of function. Spinal curvature (kyphosis or scoliosis) is not infrequently present and there may also be clubbing and redness of the end of the nose. Aside from the changes in the extremities there are no other symptoms.

In speaking of simple clubbing it was pointed out that the deformity occasionally disappeared, as for instance, following the draining of an empyema. In such cases the enlargement is confined to the soft tissues. In hypertrophic pulmonary osteo-arthropathy the bony changes are permanent. These changes may occur very rapidly, or they may develop slowly or, as is probably true of most of the cases, the changes pursue an irregular clinical course which is subject to exacerbations and remissions depending on an increased activity or improvement of the primary condition.

Relation of Simple Clubbing to Hypertrophic Pulmonary Osteo-arthropathy.—Some years ago I expressed the opinion that simple clubbing was to be looked upon as an incipient or arrested stage of hypertrophic pulmonary osteo-arthropathy, a view which Locke supports. There are several reasons for this: (1) Both simple clubbing and hypertrophic osteo-arthropathy are associated with the same general group of diseases; (2) clubbing of the fingers is one of the characteristic features of hypertrophic osteo-arthropathy; (3) the clubbing which occurs in the hippocratic fingers is identical with that occurring in osteo-arthropathy, except perhaps in the degree of development; (4) osseous changes sometimes occur in simple clubbing which are precisely the same as those seen in hypertrophic osteo-arthropathy; 12 out of 39 cases of simple clubbing studied by Locke showed bony changes.

Diagnosis.—The diagnosis is, as a rule, easy and rests on the characteristic changes in the extremities associated most frequently with disease of the lungs. The extent of the change is easily determined by Roentgen-ray studies. The condition has a superficial resemblance to *acromegaly*. The latter is a primary disease due to alterations in the pituitary body, and is ultimately fatal. While it causes enlargement of the feet and hands, the nails are not changed, and it involves the whole part symmetrically; joint effusions are rare. Furthermore, in acromegaly, the face, lower jaw, tongue and genitals are enlarged as are also the cartilages of the ear, nose, eyelids and epiglottis.

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[1] *Penna. Med. Jour.*, August, 1907.

CHAPTER XXIII

DISEASES OF THE PLEURA

PLEURISY

Etiology.—Inflammation of the pleura is a very common affection, which may manifest itself either as a primary or a secondary process. In the great majority of instances pleuritis is secondary to some preëxisting condition.

Until within recent years primary pleurisy was looked upon as of frequent occurrence. This was due to the fact that a large number of instances were observed in individuals who were apparently healthy, and in whom the attack followed exposure to cold, the so-called pleurisy *a frigore*. While a primary pleurisy can be produced by exposure to cold, it is now known, as I shall show later, that the majority of these so-called idiopathic cases are in reality tuberculous in nature.

Traumatic injury of the chest wall is the only other condition which is apt to produce a primary inflammation of the pleura.

Secondary pleurisy as a rule, originates in one of two ways: (1) By extension of an inflammatory process, involving the lungs, pericardium or mediastinum, and occasionally the liver; (2) the pleura may be infected by way of the blood stream in cases of general septicemia. Pleurisy also occurs in certain of the chronic diseases such as nephritis and gout.

By far the commonest source of pleural inflammation is pulmonary disease. There are very few instances of disease of the lung, either acute or chronic, in which the pleura is not secondarily involved. Thus we see pleurisy associated with croupous pneumonia, broncho-pneumonia, pulmonary abscess and gangrene, hemorrhagic infarction and emboli, the mycotic infections, typhoid fever and most important of all pulmonary tuberculosis.

Inasmuch as *tuberculosis* is by far the most frequent pulmonary infection encountered this disease overshadows all others as an exciting cause of pleurisy. I have already alluded to the fact that at one time idiopathic pleurisy was considered to be of frequent occurrence. But the intensive studies which have been made of tuberculosis in all its manifestations, during the past decade, have shown conclusively that the great majority of all cases of apparently primary pleurisy are tuberculous. It must never be lost sight of that primary pleurisy is relatively infrequent, and that in the absence of a demonstrable lesion which might cause it, a tuberculous origin is to be thought of whether exposure to cold has occurred or not. Hemoptysis or an attack of pleurisy are two of the ways in which a pulmonary tuberculosis may suddenly manifest itself in an individual who is apparently healthy. In most cases a readily demonstrable pulmonary lesion is present; in others the lungs seem to be normal. In every instance, however, the patient should be given the benefit of the doubt, and warned as to the possible significance of the pleurisy. In the case of a serofibrinous effusion, especially one which has developed insidiously and with no symptoms, the probability of its being tuberculous in origin is almost a certainty.

The argument is frequently used that many individuals who have had pleurisy with or without effusion, have never developed clinical tuberculosis. Of this there is no doubt. On the other hand, many more such patients actually do have tuberculosis or subsequently develop it. The records of the Phipps Institute show that of 5895 patients in whom a diagnosis of tuberculosis was made, 23.8 per cent. gave an antecedent history of pleurisy. In an analysis of two series of cases of pulmonary tuberculosis Pierce[1] found that in the first group of 1767 cases 35 per cent. gave a history of pleurisy and in the second group of 518 cases 52 per cent. gave such a history.

So long as there is doubt as to which are really primary and which are secondary to a latent tuberculosis it is unwise to assure the patient that the trouble is trivial in nature.

Morbid Anatomy.—Inflammatory changes involving the pleura may be unilateral or bilateral and may be limited to a small circumscribed area, or involve all of one side. Any part of the pleural surface may be the seat of the inflammatory process. Post-mortem experience shows that the pleural covering of the apices of the lungs, especially posteriorly, is most frequently involved, although there is little evidence of this during life (see Fig. 237). Probably the next most common site is the lower part of the chest anteriorly and laterally. It is a matter of clinical observation that pain and friction rubs are more frequently encountered in these areas than elsewhere. I have already pointed out the probable tuberculous origin of many of these cases. White[2] in an analysis of 197 autopsied cases of tuberculosis found that a favorite site for old pleuritic adhesions was along the course of the third, fourth and fifth ribs anteriorly, from the parasternal or mid-clavicular line to the mid-axillary line. In tuberculosis cases it happens not infrequently, that with disease at one apex a dry or fibrinous pleurisy will develop over the base of the opposite lung. In rare instances the pain is manifest on the side not affected. Finally, it should be mentioned that the inflammation may be limited in part or entirely to the diaphragmatic surface.

The exciting cause of the inflammation, in the great majority of cases, is either the tubercle bacillus, the pneumococcus or the streptococcus. The intensity of the inflammation depends, to some extent, on the character of the infection. In the very severe and acute attacks any one of the above-mentioned organisms may be the cause, but if the attack is insidious and attended with little or no pain the tubercle bacillus is in all probability the exciting cause.

In *fibrinous or plastic pleurisy* the surfaces, first of the visceral and then of the parietal layer, lose their luster and polished appearance, become congested and dull and finally show a fibrinous exudate which in pneumonic pleurisies is usually very widespread.

The fibrinous form may terminate as such leaving no evidence of having existed. More commonly the fibrinous deposit undergoes pro[illegible]rative changes which produce adhesions between the visceral and parietal layers. The adhesions may be extensive on both sides and may be dense in some places and fragile and easily broken up in others. Complete obliteration of the pleural sac is relatively common. In such cases the pleural surfaces are firmly united, attaching the lung to the chest wall and diaphragm throughout. Often the lung is firmly bound down and cannot be removed from the chest except by cutting it free. Chronic obliterative pleurisy may originate from an acute pneumonic pleurisy or pleurisy of other bacterial origin. In tuberculosis the pleural thickening is rarely uniform throughout. Usually it is dense at the apices while in the inferior parts the adhesions are lace-like in character. Complete obliteration of both pleural sacs is sometimes noted.

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[1] *Northwest Medicine*, vol. xvi, No. 3.

[2] Phipps Institute Report, 1907.

In a certain proportion of cases the fibrinous exudate becomes excessive in amount and in addition there is an exudate from the capillaries of the pleura, constituting what is known as *serous or serofibrinous pleurisy.* The quantity of fluid varies from that which barely can be recognized by physical signs to an amount that nearly fills the pleural sac. If the primary inflammation is severe in character the serofibrinous exudate may be poured out rapidly and evidences of an effusion be manifest within a few days from the onset of the pain. In other instances the fluid accumulates gradually and is unsuspected until it has reached a large amount. Although not a common occurrence a serous effusion is occasionally encysted (see Figs. 334 and 335).

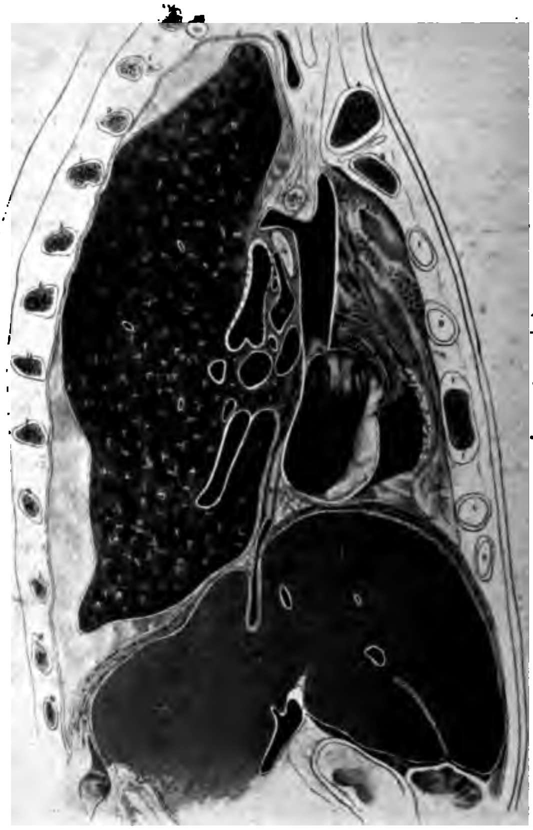

FIG. 334. Section through the right sterno-clavicular articulation, showing pneumonia (gray hepatization) with a moderate amount of serous exudate in the upper and lower margins of the pleura. (*Pirogoff.*)

The fluid in the serofibrinous form is yellowish or of a faintly green tint containing flakes or coagulated masses of fibrin. In some instances the fibrin may be so abundant that the effusion presents a gelatinous appearance. Microscopically leukocytes and a few blood cells are noted. An excess of lymphocytes points to a tuberculous origin of the disease.

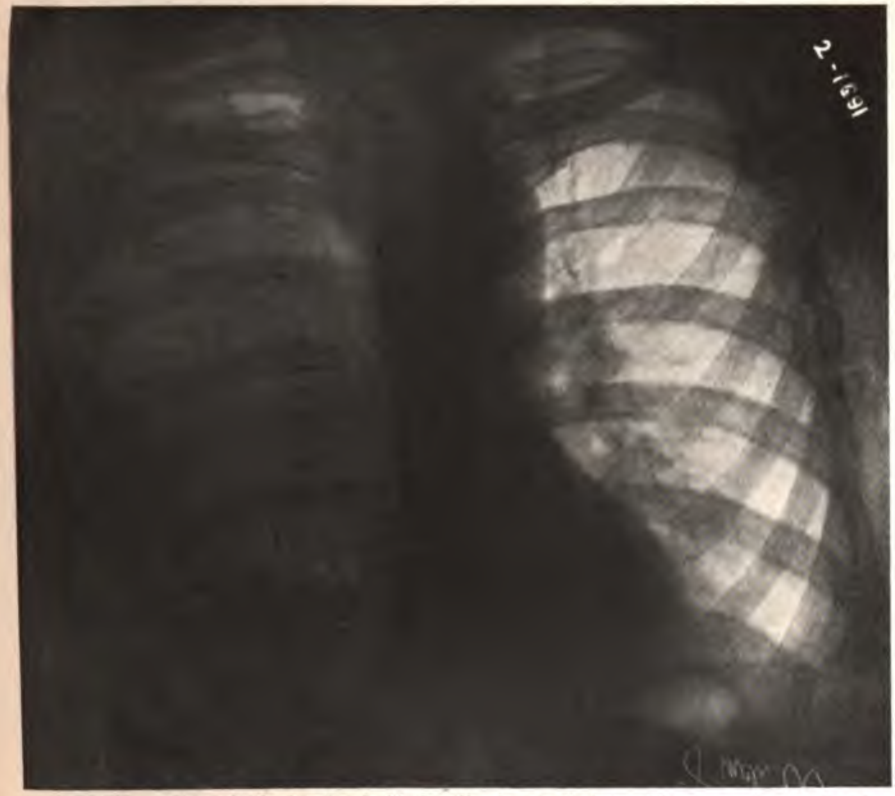

FIG. 335.—Pleural effusion (probably localized posteriorly). Fluoroscopic examination of chest: The right diaphragm was almost fixed, scarcely any movement being perceptible. Almost the entire right side of the chest was very much obscured by what appeared to be a localized effusion. This extended from the outer corner of the diaphragm upward to the apex, but did not extend to the heart and aortic shadows, over one-quarter to three-quarters of an inch of clear space being seen between the opaque region and the aortic and heart shadows. The shading was not so dense as to hide the ribs and diaphragm. On lateral inspection a clear space could be seen in front of the chest, which would indicate that the possible effusion was posterior. (*Dr. D. R. Bowen.*)

In long-standing cases of serous effusions which have been largely absorbed the occurrence of *cholesterol* in the fluid has been noted. Two such cases have been reported by Schulman[1] and Arnell.[2] For details as to the bacteriology of pleural exudates the reader is referred to any of the standard text-books on laboratory diagnosis.

Artificial pneumothorax in the treatment of pulmonary tuberculosis is very frequently followed by an effusion which may require frequent tappings. Occasionally the effusion becomes purulent; two such accidents occurred among the cases treated at the Phipps Institute.

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[1] *Jour. Amer. Med. Assoc.*, April 28, 1917.

[2] *Hygiea*, Aug. 16, lxxix, No. 5.

Purulent effusions may be such from the onset and this is especially true when the offending organism is the streptococcus. It should be borne in mind that in pneumococcus pleurisies the exudate is usually purulent. More frequently, however, the effusion is primarily serous in character, or possibly turbid in appearance. The subject of empyema will be dealt with under a separate heading.

At times the effusion is hemorrhagic in character. Hemorrhagic effusions and hemothorax are also considered under separate headings.

Pressure Effects.—The degree of pressure which will be exerted on the lung of the affected side and adjacent viscera will depend on the size of the effusion. Even with very small collections of fluid the base of the lung in immediate contact with the fluid becomes atelectatic. As the fluid accumulates the lung is more and more compressed until, in extreme cases, it has been reduced to a small, elongated and flattened mass occupying the upper and posterior part of the chest along the spine (see Fig. 344). The lung is generally airless and tough and leathery to the touch. If the effusion occupies the right side of the chest the heart is displaced to the left beyond its normal position, and the liver is forced downward by the flattening of the diaphragm (see Fig. 346). If the effusion is left-sided the heart is displaced to the right; in extreme cases pulsation may be seen in the fourth or fifth interspace to the right of the sternum. The diaphragm is also flattened on this side forcing downward the spleen, stomach and left lobe of the liver (see Fig. 103).

If the effusion develops rapidly dyspnea may occur, because the opposite lung has not had time to adjust itself to the increased work thrust upon it. In slowly developing effusions considerable compensatory emphysema will be noted in the unaffected lung. Léon, LePlay and Mantoux[1] have shown experimentally that after establishing a complete unilateral thorax in dogs a partially progressive increase in the thorax is established on the opposite side. In order to determine how small a portion of functionating lung was compatible with life they found that one-sixth of the total lung capacity was sufficient to maintain life under ordinary conditions. This shows the extent to which the lungs may be functionally incapacitated either as the result of pleural effusion or extensive tuberculous disease, without producing death.

From the foregoing description it will be seen that the various stages have a fixed sequence, a fact of the greatest importance, because they correspond in a striking manner to the physical signs and even the symptoms of the disease. To recapitulate: (1) The dry stage in which the inflamed pleural surface becomes vascular and loses its moist and polished appearance. (2) The fibrinous stage in which the affected area becomes coated with flakes of lymph. It is, of course, understood that the process in the majority of instances does not progress beyond the first or second stages. Chronic obliterative pleurisy is probably a sequel of a widespread fibrinous deposit which undergoes connective tissue changes, and firmly unites the two layers of the pleura. (3) The stage of effusion which may be serous, serofibrinous, purulent or hemorrhagic, and which appears first at the base of the chest. As the accumulation of fluid increases pressure effects manifest themselves by compression of th lung and displacement of the viscera. (4) The stage of absorption and reëxpansion of the lung. Whether the lung will return to its normal taste will depend largely on the length of time it has been subjected to compression. If the lung has been compressed for a long time or becomes adherent to the chest wall in its collapsed condition, extensive pulmonary fibrosis is a probable sequence. Another factor which often prevents the lung from being restored to its normal condition is fixation of the diaphragm (see section on Diaphragmatis).

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[1] *Jour. de physiol. et de pathol. gen.*, 15, S. 16–22, 1913.

Symptoms.—Acute pleurisy may be ushered in with a chill or mere chilliness, followed by fever. In the majority of the cases, however, the first intimation of trouble is *pain* in the side. The character of the pain is extremely variable. Involvement of the apical pleura rarely causes a sharp pain but as a rule manifests itself by a sense of soreness or pain in the shoulder. The latter is often regarded as rheumatic. On the other hand, it may be agonizing in character and every movement or inspiratory effort intensifies the stabbing, lancinating quality of the pain. The pain is usually referred to the nipple or axillary regions, but may be noted beneath the scapula. If the diaphragmatic area is involved the pain may be felt low down in the back, in the abdomen, thus simulating acute appendicitis, etc., or in the neck (see p. 580). The pain is, as a rule, more intense during inspiration than expiration, but in rare instances is felt solely with the latter act. The pain is nearly always aggravated by cough. The *respirations* are shallow and rapid in dry pleurisy if the pain is severe, but breathing of this type is to be distinguished from true dyspnea. The patient by breathing in this rapid manner unconsciously seeks to prevent the inflamed pleural surfaces from rubbing together any more than possible; it is probable also that the increased frequency of the respirations is compensatory. If *cough* is present it is usually short and hacking in character, for the same reason that the respiratory act is hurried, namely, the effort to keep the inflamed surfaces apart. The cough is usually unattended by expectoration, but if the latter is present it is scanty, and consists simply of mucus.

If the pleurisy is at all severe it is usually attended by *fever* up to 102 or 103°F. It may be continuous from the start, or slightly remittent in character (Fig. 336). As the acute inflammatory condition subsides, the fever falls gradually by lysis. In the protracted cases the temperature tends to become more markedly remittent and assumes the form of hectic fever.

The *pulse* rate is nearly always increased to 100 and over and irregularity of the pulse is not infrequent.

The general symptoms are those of an acute illness, namely, general malaise, mental dulness and headache, especially if the fever is high, pallor, a distaste for food and constipation.

With the development of an *effusion* the pain disappears entirely or at most there is a sensation of soreness which may be elicited only by pressure of the fingers over the site of the inflamed area. If the respirations are hurried in this stage of the disease, they are due to a true dyspnea owing to compression of the lung. If, however, the effusion has not been preceded by pain and has developed insidiously, dyspnea may be absent or noticed only on unusual exertion. Effusions of this type often fill the pleural sac without attracting attention. This is due to the fact that the lung on the unaffected side has had time to accommodate itself to the increased work put upon it. Under such circumstances the patient seeks relief because he feels that he is gradually losing health, but is unable to localize his trouble. His symptoms are as a rule general, such as malaise, weakness and some perversion of the gastro-intestinal functions. If the effusion has persisted for some time, loss of weight is usually a prominent symptom.

It has been noted also that with the development of an effusion the urinary secretion is commonly reduced in amount and that the urine is concentrated and of a high specific gravity. With absorption of the fluid the urine becomes increased in amount.

Purulent effusions will be considered under a separate heading.

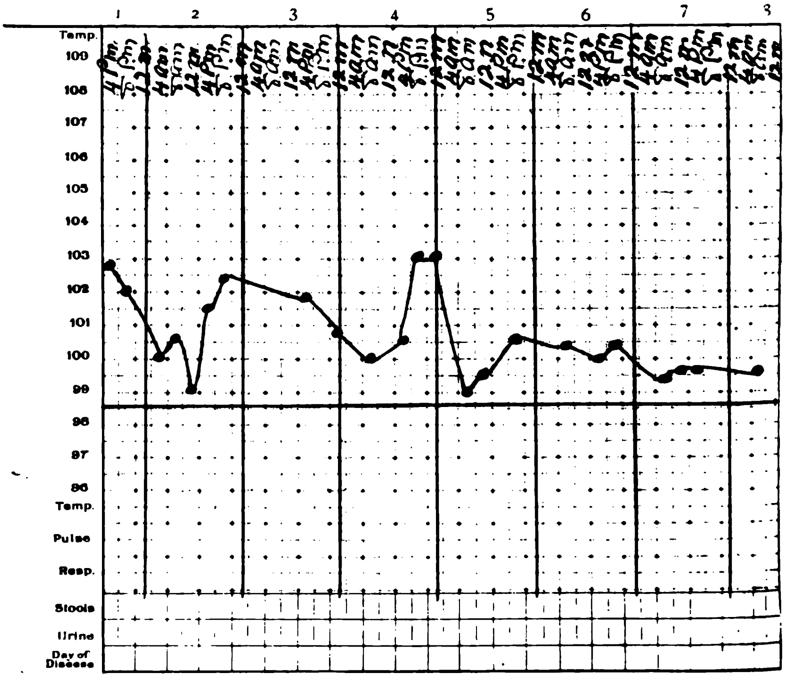

FIG. 336.—Acute tuberculous pleurisy.

Physical Signs.—In the great majority of cases our attention is directed to the pleura, because of the location of the pain. Examination will show that the pleural inflammation is either the result of some obvious pulmonary affection, or that the pleurisy is either primary or secondary to pulmonary disease which is masked, as for instance, tuberculosis. On the other hand, definite signs of pleural inflammation may be present in some cases without the presence of pain or other symptoms, the finding being purely accidental. In still other instances well-marked pain indicative of pleural inflammation may not be associated with any physical signs whatsoever.

DRY PLEURISY.—*Inspection.*—If the pain is at all severe objective signs are usually present. The breathing is shallow and hurried. The

may lie on the sound side, or sit up and be inclined toward the
l side with the elbow pressed against the chest wall. Change of
intensifies the pain. Examination of the chest shows restriction
xpansion on the affected side.

pation.—A friction fremitus may be felt, but this is rarely so.
atient is examined from behind and a hand is placed on each side
hest wall, it will be noted that the inspiratory excursion is much
hed on the affected side. If firm pressure is made with the hands,
lief is experienced by the patient as the inspiratory excursion is
stricted. It is because of this that adhesive strips are applied to
cted side in order to diminish the pain.

the comparatively rare instances in which inflammation of the
exists without one being able to hear a friction rub, its presence
established by firm pressure with the finger tips over the site
pain. This procedure will often reveal distinct tenderness. It
to be noted that in those cases in which a friction rub is accident-
covered that some tenderness on pressure can usually be elicited.

cussion.—There are no alterations in the percussion note during
ge.

cultation.—The presence of an audible friction rub is the charac-
feature of an inflamed pleura. The friction rub may be heard
wide area, or it may be localized in a spot no larger than that cov-
the bell of the stethoscope.

friction rub becomes audible as the inflamed surfaces of the
l and parietal layers pass over each other during the respiratory
n the majority of cases it is heard only during the inspiratory
nd is usually most pronounced just as inspiration ceases. It may
rd, however, during both inspiration and expiration and rarely
occurs during expiration only.

friction rub suggests a rubbing or grazing sound; it may be rough
ating in character, or it may resemble the sound produced by
g leather.

friction rub is usually a very local sound and is always best heard
e parts where the respiratory excursion is greatest, namely, the
art of the chest below the nipple, axilla or about the angle of the
. I have already pointed out that the area beneath the nipple
illa is frequently the site of pleural adhesions. The most com-
ite of pleural adhesions is over the apex posteriorly. Severe
ic pain is rarely experienced in this region, however, owing to
ry limited movement of the apices of the lungs. Pain due to
pleurisy usually simulates rheumatic pain in the shoulder, or
ar soreness, and is a common symptom of apical tuberculosis.
ss or tenderness beneath the clavicle is a frequent occurrence in
ary tuberculosis, especially if cavity formation has taken place.

RINOUS PLEURISY.—The physical signs during the second stage
inflammatory process differ but little from those just described.
ly points of difference lie in the fact that dry pleurisy, in which
mbrane is simply roughened, is chiefly tuberculous in origin and
pt to involve a large area of the pleural surface. Fibrinous pleu-
the other hand, while it may be tuberculous in origin is seen in its
ypical form when associated with an acute inflammatory condition
lung, particularly croupous pneumonia. When associated with

pneumonia the exudate may spread over the visceral layer of an entire lobe of the lung. This explains why one can occasionally detect some impairment of resonance over a lung which a day or so later shows signs of a croupous pneumonia. Furthermore, a widespread fibrinous deposit will manifest itself by the presence of a friction rub heard over a wider area than is usually encountered in simple dry pleurisy. The condition is usually limited to one side but it may be bilateral.

DIAPHRAGMATIC PLEURISY.—This form of pleurisy is deserving of special mention because it may simulate an acute abdominal condition. The diaphragmatic pleura may become inflamed as any other portion of the pleural surface or it may be secondarily involved from the abdomen. The right side is involved about twice as often as the left.

In inflammation of the diaphragmatic pleura, the subjective symptoms, as a rule, are out of all proportion to the objective signs. Both because the lungs have the widest respiratory excursion at the base and because of the part taken by the diaphragm in the respiratory act, pleural inflammation in this situation gives rise to the most intense pain (see Fig. 41).

The characteristic feature of the pain is that it is referred to parts distant from the seat of the inflammation. The referred pain of diaphragmatic pleurisy is due to involvement of the spinal nerves supplying the abdominal wall. The pain is, therefore, superficial and not deep-seated as in visceral pain. In cases of diaphragmatic pleurisy or subphrenic abscess, steady, deep pressure over the gall-bladder or appendix is well borne. If, however, these organs are the seat of inflammatory changes the deep pressure gives rise to severe pain.

Capps[1] in a study of 61 cases of diaphragmatic pleurisy found the pain referred to the gall-bladder 10 times; lateral to and slightly above the navel 15 times; and other points, including the lumbar region, in the remainder. If diaphragmatic pleurisy is suspected, search should be made for tender points in the neck and shoulder region as referred pain to these points is commonly present.

In addition to the pain there is usually some cough, expectoration, rapid respirations and a leukocytosis. Herpes may be present.

PLEURAL EFFUSION.—*Inspection.*—Unless the fluid has accumulated rapidly dyspnea will not be noted, otherwise the breathing may be hurried. Cyanosis is rarely seen even when the exudation fills the pleural sac in a brief space of time. In common with many other inflammatory conditions within the thorax inequality of the pupils is not infrequent, the larger pupil being on the same side as the thoracic trouble.

Turning our attention to the chest itself the first thing to be noted is the respiratory movements. If one side is seen to expand less than its fellow the lack of expansion may be due to one of two things, an acute or chronic inflammatory process (usually the latter), of the lung, or fluid in the pleural sac. If the lung itself is the cause, there is usually some retraction of the affected side, especially if the process is chronic in nature. In the case of effusion the affected side appears larger and the intercostal spaces are filled out, or even present a bulging appearance. The difference in the two sides of the chest can be shown also by means of a cyrtometer. Litten's sign is absent in pleural effusions.

Next we have to consider the position of the apex beat of the heart

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[1] *Am. Jour. Med. Sci.*, March, 1916.

It is to be borne in mind that displacement of the viscera constitutes one of the most important pieces of evidence as to whether a pleural effusion is present or not. If the apex beat is displaced to the left it may be aused by one of three things: (1) It may be due to hypertrophy or dilaation of the heart itself. It is hardly likely that this would cause any onfusion, because of other associated cardiac signs. (2) The heart may e pulled to the left as the result of a fibrosis involving the left lung. In uch a case, however, it will be noted that the left chest is retracted. 3) It may be due to a pleural effusion on the right side which has dislaced the heart to the left. If the heart is displaced to the right it is he result of one of two things: (1) A chronic fibroid condition of the ight lung which has drawn the heart to the right; or (2) a pleural ffusion on the left side. The apex beat may not be visible either beause it has been displaced to the right and lies behind the sternum, or because the chest wall is so thick the impulse cannot be seen. Under these circumstances it may be located by palpation or auscultation.

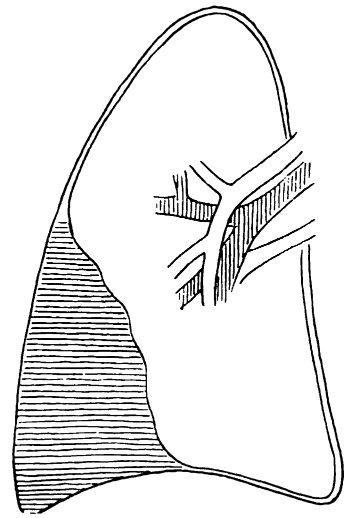


FIG. 337.—The first effect of a small effusion is to push the lung forward. Physical signs are confined to the posterior aspect of chest and there is no displacement of the viscera.

Palpation.—If there is any doubt as to whether the two lungs expand equally or not, one can almost invariably arrive at a correct conclusion by placing the hands on corresponding parts of the two sides of the chest. In this way inequalities of expansion can be felt when they are not readily detected by the eye.

Tactile fremitus is absent over the affected side unless the underlying ung be solidified (see Fig. 57). If the apex beat of the heart is not isible on inspection it may be located by palpation.

Percussion.—In case the effusion is small in amount it is important o determine the lower limit of pulmonary resonance. This is on a level ith the tenth dorsal vertebra. If it is found to be an inch or two higher n one side it points to a small effusion if there has been a recent history pain on that side. An effusion is not apt to be suspected, however, nless it has reached a line corresponding with the angle of the scapula.

The percussion note over an effusion is flat and being one of the varieties of dulness is high-pitched. Furthermore, there is a sensation of resistance imparted to the finger acting as the plexor.

In determining the extent of the effusion the percussion should be performed lightly so as to distinguish between the absolute dulness or flatness over the fluid and the relative dulness over the lower part of the lung which is collapsed. The percussion should be performed at right angles to the spinal column and at different levels, as it has long been recognized that the fluid extends higher in one portion of the chest than in others. As the fluid accumulates in the pleural sac the lung is pushed forward as seen in Figs. 337 and 338. In these small effusions the dulness is highest at the spine and confined to the back, rarely extending anterior to the posterior axillary line. In small effusions displacement of the viscera is not apt to occur. It should be borne in mind that small effusions may exist without their being physically demonstrable. Experimentally (post-mortem), 400 c.c. (℥ xiii) of fluid in the pleural cavity of the adult, and 120 c.c. in a child of twelve can be demonstrated by physical signs. Clinically, even smaller amounts may be detected, but small effusions may exist and may be visible fluoroscopically as a flattening of the diaphragm which cannot be demonstrated by physical signs.

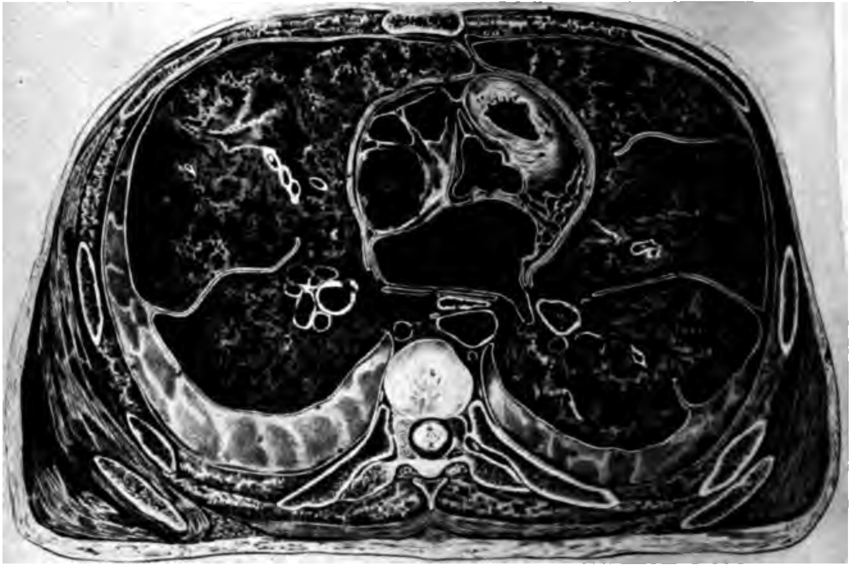


FIG. 338.—Transverse section through the nipple line.

Diffuse pulmonary tuberculosis in a man aged 25 years, with a bilateral serofibrinous effusion which has accumulated posteriorly. The anterior pulmonary margins show vicarious emphysema and completely overlap the heart, which is distended with blood clots. (*Pirogoff.*)

In moderate effusions the lowest level of dulness is at the spine; it then rises toward the middle of the scapula and slopes downward, assuming the shape of the letter S as it passes toward the front. As the effusion increases in size the lung is floated and pushed upward and backward toward the spine and while the lower part of the lung is relaxed. As a lt of this the note over the relaxed portion of the lung is not dull has a tympanitic quality, sometimes referred to as Skodaic tympany koda's resonance. The area in which this note is elicited is shown in 339. This space is known as *Garland's triangle.*

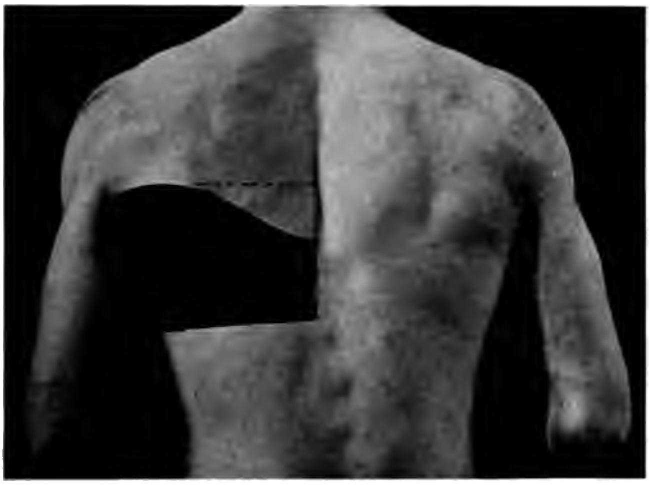


Fig. 339.—Showing area skodaic tympany (Garland's triangle).

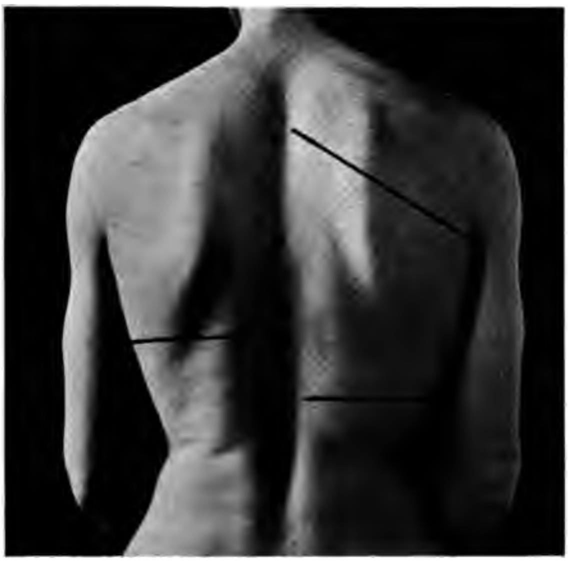


Fig. 340.—Upper and lower limits of dulness in a massive effusion.

n massive effusions the highest point of dulness is posteriorly at the (see Fig. 340) and lowest in the front of the chest. Over the col- d portion of the lung the note may be tympanitic in character but lung has been so completely compressed as to be devoid of air the note will be dull. In massive effusions the flat or dull percussion note anteriorly may be elicited some distance to the left or right (as the case may be) of the affected side (see Figs. 341 and 342).

By the time the effusion has reached any considerable amount displacement of the viscera will be noted. On the right side the weight of the fluid flattens the diaphragm and as a result the liver is forced downward. This may be noted by palpation or percussion; usually the latter. On the left side the filling of the complementary pleural space and the flattening of the diaphragm sometimes causes a disappearance of the tympanitic area in the anterior and lower part of the chest. This tympanitic area is known as Traube's space (see Fig. 89). Dulness over this space is met with, as a rule, in massive effusions only and even then may not occur as the complementary pleural space is often obliterated by firm adhesions. Massive effusion on the left side, particularly empyemas, may force the diaphragm downward to such an extent that the fluid bulges below the costal margin. This may be mistaken for an abdominal tumor.

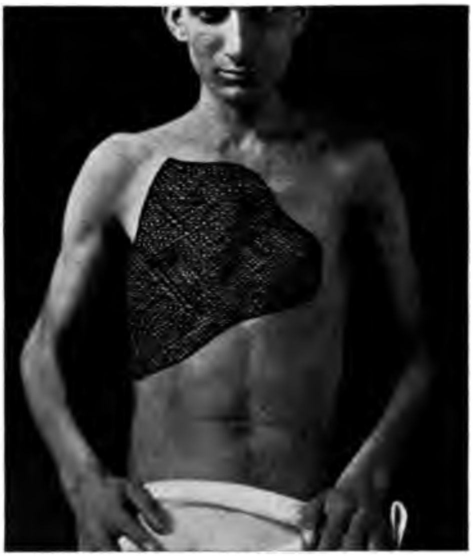
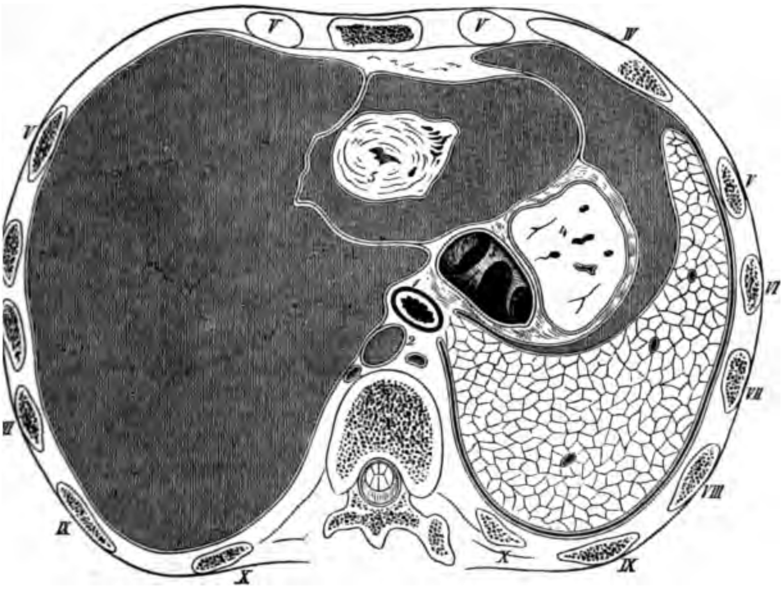


FIG. 341.—Area of percussion dulness in a massive pleural effusion. The dulness extends beyond the middle line and merges into cardiac dulness.

Movable dulness, when present, is a valuable sign. As a rule, it is obtained in moderate-sized effusions only. If the effusion extends in front as high as the fourth interspace when the patient is sitting up, it will be found that when he lies down the upper limit of dulness is in the interspace below. This is due to the fluid gravitating to the most dependent portion of the chest, namely, the back. Movable dulness cannot be demonstrated in massive effusions extending to or above the second rib in front nor in small effusions when the signs are confined entirely to the back. Neither can it be demonstrated in moderate-sized effusions if the lung has become adherent. Movable dulness on change of posture, when it can be demonstrated, is the most pathognomonic physical sign. As a general rule, however, except in cases of hydropneumothorax, mobility of dulness is not readily demonstrable and con-able time is often required before the fluid gravitates to the lower on of the pleural cavity.

ı sign of some value is the paravertebral triangle of dulness, somes referred to as *Grocco's triangle.* This triangle consists of an area lative percussion dulness on the side opposite the effusion. The cal line of the triangle extends along the middle of the spinal column a point slightly above the upper level of the effusion to the tenth al vertebra, the latter representing the lower limit of pulmonary resice; the base of the triangle extends outward along the lower limit ırmal pulmonary resonance for a distance varying from 1 to 3 inches; hypothenuse joins these two lines (see Fig. 343). The dulness is



G. 342.—Left-sided hydrothorax and hydropericardium. 1, Esophagus. 2, Aorta. erior vena cava. 4, Liver. 5, Heart.
ıe cardiac apex is displaced backward and to the right. The left pleural sac extends ıorly to the mid-sternal line, and posteriorly even beyond it. (*After Pirogoff.*)

marked over the spine. There is some doubt as to how this ıgular area of dulness is produced. The generally accepted view, ever, is that the contents of the posterior mediastinum are displaced Fig. 342). The chief value of this sign is in differentiating between ral effusions and tumors involving the lung or pleura; if present, it ıt likely that a tumor exists.

lusculation.—The characteristic friction rub heard at the base of chest in the dry and fibrinous stage disappears as the two pleural ıces are separated by the effusion. A friction rub may be heard, ever, just above the fluid in the axilla or anterior and superior porof the chest where the inflamed pleural surfaces are still in contact. the area occupied by the effusion the breath sounds may be weakly ular or bronchial in character. As the fluid increases in amount ıreath sounds tend to become more and more distant and in massive effusions may be entirely absent. In a certain proportion of cases, especially when the effusion is large, loud bronchial breathing may be heard. For the explanation of this phenomenon see page 61.

In addition to the friction rub heard above the level of the effusion, râles produced within the lung may be heard in the same areas. The pulmonary râles may be due to congestion in the collapsed portion of the lung or they may be due to the separation of the walls of the finer bronchioles and alveoli in the atelectatic portion of the lung. The breath sounds above the effusion are usually of a harsh broncho-vesicular character.

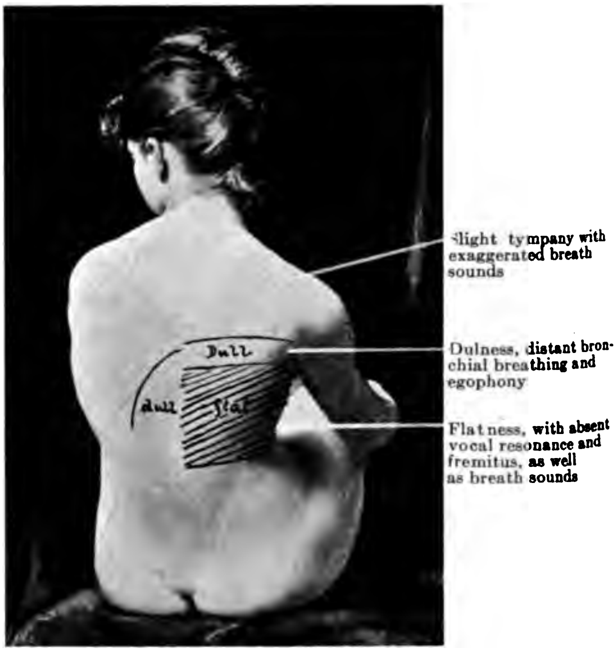

Slight tympany with exaggerated breath sounds
Dulness, distant bronchial breathing and egophony
Flatness, with absent vocal resonance and fremitus, as well as breath sounds
Dull
dull
flat

FIG. 343.—Grocco's triangle.

On auscultation of the voice sounds, the same phenomena are encountered as in auscultation of the breath sounds. The spoken voice may have a distant sound or it may be distinctly bronchophonic. Just as in the case of the breath sounds a very large accumulation of fluid may cause complete disappearance of the voice sounds. The presence of egophony, while not a constant finding, occurs with great frequency in association with pleural effusion.

In some instances auscultation of the whispered voice is of service in distinguishing between serous and purulent effusions. If the effusion is serous, one will be able to hear, at times, the whispered voice transmitted through the fluid but if the effusion is purulent no such transmission occurs. This sign, known as Baccelli's, is, however, of relative little value.

In not a few instances the question of whether the heart is displaced or not will depend on the use of the stethoscope. In spite of the [illegible] lacement of the heart which sometimes occurs, the cardiac sounds are ally normal.

Exploratory Puncture.—No matter how positive the evidence is as he presence of fluid in the pleural cavity the use of an exploratory dle should never be omitted. There are two reasons for this. In first place the actual demonstration of fluid by means of the needle ches the diagnosis. In the second place our duty to the patient does end with the discovery of the presence of fluid nor is our work comted until the character of fluid has been demonstrated. Inasmuch as can never tell, with any degree of certainty, whether the fluid is ous or purulent, the needle must be resorted to. If purulent fluid is hdrawn, immediate drainage of the pleural cavity is mandatory, if patient is to escape the horrors of a chronic empyema.

In making an exploratory puncture of the pleura or lung the areas which the viscera are located should be kept in mind. In the great ority of instances when fluid is suspected the exploring needle is rted in an interspace just below or slightly anteriorly to the angle of scapula. If too fine a needle is used a dry tap may result if the fluid sists of thick pus. For this reason the needle should have a caliber t least 2 mm.

Sterilization of the skin with tincture of iodine and the use of a sterile dle renders the procedure entirely harmless. Even if fluid is not ent and the needle punctures the lung, no harm will result providing rules for cleanliness have been observed.

The X-rays are helpful as a diagnostic measure, especially in case the sion is loculated.

Physical Signs Following Absorption of the Exudate.—Even in those s in which the effusion exists but a relatively short time the lung s not expand immediately to its former state following removal of fluid. If the effusion has existed for several weeks before it is disered and removed, or if it is allowed purposely to remain and undergo ntaneous absorption, it may take several months for the lung to bee completely reëxpanded. Another and perhaps a more important tor in reducing the functional capacity of the lung, is fixation of the phragm on the affected side. If, therefore, the case is seen after abption of the fluid and before the lung has reëxpanded confusion may se unless the morbid anatomy and the physical signs associated with is phase of the process are kept in mind (see p. 642).

If the effusion has followed an attack of dry pleurisy the lung will ually reëxpand without the formation of pleuritic adhesions or at the ost the adhesions will be slight.

If a fibrinous exudate has been the starting point of the effusion and ecially if the fibrinous deposit has been extensive the two pleural faces may become firmly adherent following absorption of the fluid then prevent complete reëxpansion. Chronic obliterative pleurisy bably originates in this way in the majority of cases.

Following spontaneous absorption of the pleural exudate or its real by paracentesis the following physical signs will be noted:

nspection.—Expansion on the affected side will be restricted but if atient has been seen from the onset the respiratory excursion will eater than when the effusion was present. Flattening and bulging interspace will have disappeared and the apex beat of the heart e seen in its normal position.

Palpation.—Tactile fremitus may or may not be present, depending on how much the lung has expanded.

Percussion.—The percussion note will still be dull over the base of the lung posteriorly. It will be found, however, that the dull area does not conform to the shape noted when fluid was present. As the diaphragm is relieved of the weight of the fluid, the liver is no longer depressed and the tympanitic note over Traube's space returns. Tympany may replace dulness elsewhere.

Auscultation.—At the base of the affected side the breath sounds are still absent or distant but not over such a wide area, and the same is true of the voice sounds. With the removal of the fluid a friction rub again becomes audible especially in the area about the angle of the scapula. Deep breathing may bring out fine crepitant râles as the result of forcible distention of the atelectatic portion of the lung.

This picture may remain unchanged for weeks or months. Gradually, however, unless the lung has been bound down by adhesions, the complementary pleural space obliterated or the diaphragm rendered immobile, these signs disappear and leave little or no evidence of damage. It is desirable to examine such cases with the fluoroscope as the persistence of the abnormal physical signs may be due to changes in the diaphragm.

Diagnosis.—In the dry and fibrinous stages pleurisy may be confused with intercostal neuralgia, pleurodynia and herpes zoster.

In *intercostal neuralgia* there is absence of fever and a friction rub. The pain while localized is darting and neuralgic in character and tenderness is present at the points of exit of the intercostal nerves. Occasionally these phenomena precede an attack of herpes zoster.

In *pleurodynia* the pain is confined to one side but is not so sharply localized as pleurisy and it may leave one side and affect the other. Muscular movement as well as breathing aggravates the pain. Tenderness on pressure is usually present. There is neither fever nor a friction rub.

When the pleural effusion develops rapidly and the attack is abrupt in onset with pain in the side, fever, hurried respirations, and bronchial breathing over one lung, it must be distinguished from *croupous pneumonia.* It is remarkable how many cases of acute pleurisy with effusion escape notice. A very large proportion of cases of croupous pneumonia with delayed resolution are probably of this nature. The acuteness of the attack and the bronchial breathing can very easily cause the unwary to make a mistake. Later as the effusion absorbs and abnormal physical signs persist in the base of the lung it is assumed that resolution of the pneumonia has been delayed. In this connection it might be stated that as a sequel of pneumonia, delayed resolution is a very rare occurrence.

In doubtful cases the differentiation between pleurisy with effusion and croupous pneumonia rests upon two facts. (1) Displacement of the viscera, and (2) the use of an exploring needle. In pleurisy the viscera are displaced while in croupous pneumonia they are not. If an effusion is present serous or purulent fluid is obtained with the exploring needle while if pneumonia is present a few drops of dark red blood may appear in the syringe.

Malignant disease of the lung or pleura is relatively uncommon but when it involves a lower lobe is more frequently mistaken for a pleural

an any other condition. This is due to the fact that the tumor
s an absolutely flat note devoid of any semblance of resonance
the voice and breath sounds are very distant or absent. In
e one's suspicion might be aroused by the absence of the para-
area of dulness on the opposite side. As this sign is sometimes
the presence of an effusion it cannot be relied on absolutely.
gain the displacement of the viscera and the exploring needle
ain reliance in escaping an error in diagnosis.

IC PLEURISY.—Two forms of chronic pleuritis are recognized:
tent reaccumulation of serofibrinous or purulent fluid after re-
pings or operation Reaccumulation of a serofibrinous effusion
ommon in tuberculosis and malignant disease. In addition an
hich has been allowed to remain in the chest too long shows a
ndency to reaccumulate. In a case which came under my care
effusion had gone unrecognized for four months. Although
equently the fluid rapidly reaccumulated after each operation.
ronic dry pleurisy is a common sequel of the acute fibrinous
effusions either serofibrinous or purulent. It is also very fre-
en in association with chronic pulmonary tuberculosis. Partial
n of the pleural sac is seen most often at the apices of the lung
ost constantly present in tuberculosis. Over the lower lobes
ating pleurisy is also common but the adhesions are less dense.
jority of such instances the adhesions are delicate, lace-like
broken up. Among 197 autopsies at the Phipps Institute
g pleuritis was noted 49 times over the left lower lobe and 71
the right lower lobe. Dense thickening of the pleura over the
s is usually associated with massive fibrosis of the lung and is
equel of an effusion, especially the purulent type. Chronic
sually has reference to that portion of the pleura which over-
wer lobes of the lungs.

ic thickening of the pleura is a diagnosis which is frequently
xplain restriction of motion, impairment of the percussion note
or less suppressed breath sounds at the base of the lung poste-
ersonally, I have rarely seen at the autopsy table thickening
ura sufficient to account for these signs. Even in those cases
there has been any marked degree of thickening the lung was
ved in the chronic inflammatory process. Probably the most
factor in giving rise to these physical signs, especially after
is immobility of the diaphragm. The diaphragm may have
icated in the inflammatory process or, what is not uncommon,
may have formed between the chest wall and the diaphragm
erating the phreno-costal sulcus. As a result the movement of
agm is interfered with. The effect of inflammatory conditions
phragm has been considered in detail in the section on "Diseases
phragm."

EMPYEMA

(Purulent pleurisy, pyothorax)

one of the varieties of pleuritis; but in view of certain patholog-
liarities it seems best to consider it as a distinct disease.
ore, it is usually secondary to conditions in which from the very
re is a decided tendency toward the formation of pus.

Etiology.—The organism which most frequently causes the cond is the pneumococcus. In children the percentage is very high. St tococci rank next in importance. A number of studies that have made of the bacteriology of empyemata show that from one-half to thirds of the cases are due to the pneumococcus and about one-qua to the streptococcus.

Whether the tubercle bacillus alone can cause an empyema is not cl In a certain proportion of cases tubercle bacilli can be demonstrate the fluid but usually in association with other organisms. The so-ca sterile purulent effusions are nearly always associated with a tubercul process. Occasionally the influenza bacillus is found in pure culture more rarely other bacteria.

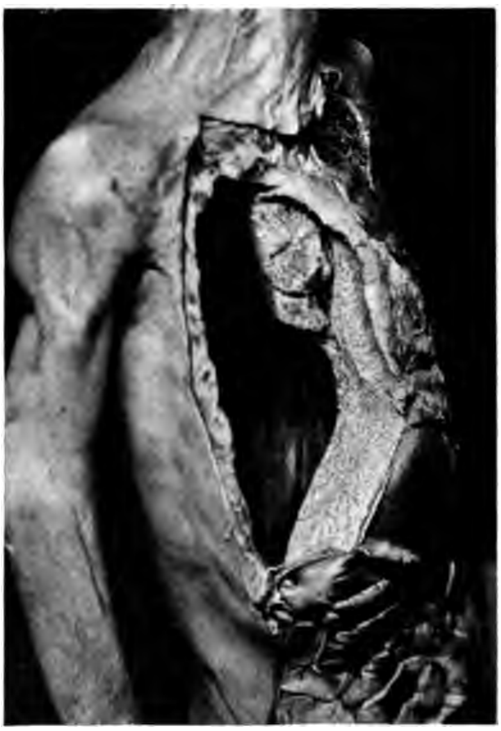


FIG. 344.—Empyema—shows the right lung which has become completely atelect
(*Courtesy of Dr. W. J. Calvert.*)

As an exciting cause of empyema, croupous pneumonia easily ra first. Empyema is also a relatively common complication of tuberculo In certain of the infectious diseases, notably scarlet fever, the condit is not infrequently met with. Empyema may arise also as the resul penetrating wounds of the chest wall or as the result of disease invol the mediastinum.

Morbid Anatomy.—In pneumococcic empyemata the effusion con of a thick creamy pus having a sweetish odor. When allowed to s the fluid separates into a clear, greenish yellow, upper layer, the cel ments sinking to the bottom. When due to the streptococcus the id may be only turbid in appearance.

An empyema is most apt to develop as the result of an extensive rinous exudate such as occurs in pneumonia where the membrane is ick and presents a shaggy appearance. In tuberculous cases the usion may be changed from a serous to a purulent one as the result of fection from the lung. The effusion may be massive, filling the tire pleural sac (see Figs. 344 and 345), or it may occupy but a portion ereof. Encapsulated effusions are considered separately.

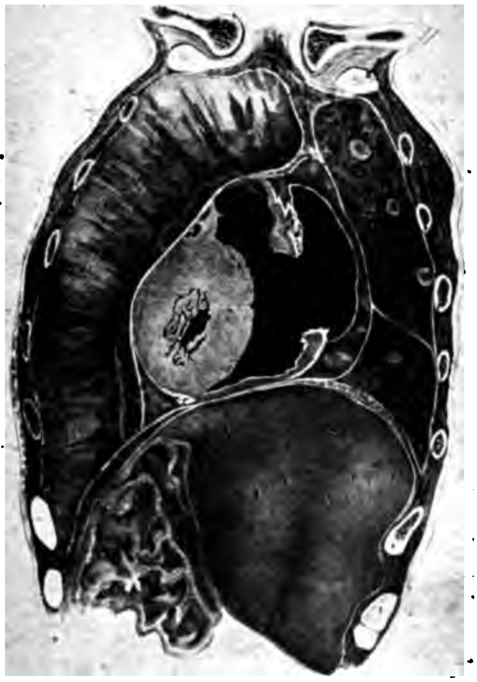


Fig. 345.—Massive left-sided empyema occupying practically the whole pleural ity. The lung is represented by two small fibrous masses adherent to the pericardium ich contains a small serous effusion. The heart, which is enlarged, is filled with blood t. The diaphragm is pushed downward and the right lung greatly compressed. rogoff.)

Unless the purulent material is removed within a short time from its set the lower portion of the lung in contact with the fluid becomes mly adherent to the chest wall and mediastinum. The adhesions be- me organized very quickly and the lung is thus firmly anchored and evented from reëxpanding even when the fluid is removed. The longer e purulent material is allowed to remain the more certain it becomes at the lung cannot be reëxpanded. If the empyema has existed for eral months the parietal pleura forming the sac becomes greatly thick- d and in places may become eroded. In moderate-sized effusions after lung has become adherent the fluid is then completely walled off and lly constitutes an abscess (see Figs. 346 and 347). The effusion may remain indefinitely the patient eventually succumbing as the result of septic absorption. In not a few cases the purulent material is gradually absorbed. Following absorption of the pus calcium salts in the form of plates are frequently deposited at the base of the cavity. In rarer instances the pus ruptures into the lung and is discharged externally through a bronchus or it may rupture externally through the chest wall—empyema necessitatis (see Fig. 348).

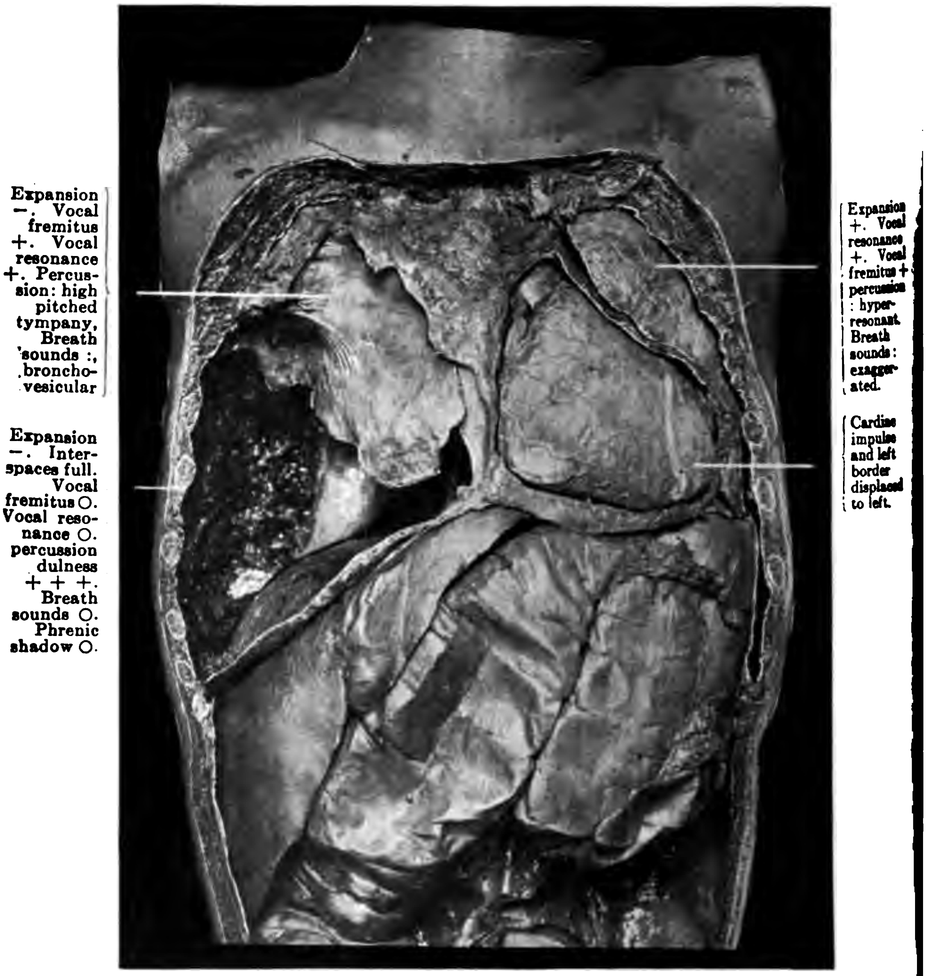
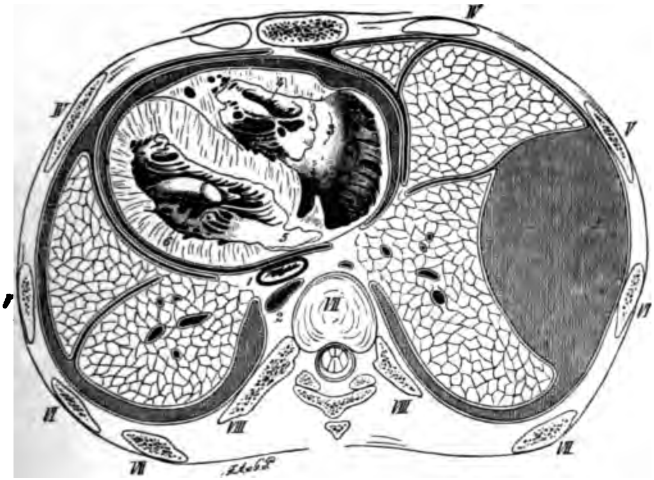

Expansion —. Vocal fremitus +. Vocal resonance +. Percussion: high pitched tympany, Breath sounds :, broncho-vesicular
Expansion —. Interspaces full. Vocal fremitus O. Vocal resonance O. percussion dulness + + +. Breath sounds O. Phrenic shadow O.
Expansion +. Vocal resonance +. Vocal fremitus + percussion : hyper-resonant. Breath sounds: exaggerated.
Cardiac impulse and left border displaced to left.

FIG. 346.—Empyema encysted in lower part of chest. Note the flattening and low position of the diaphragm and the downward displacement of the liver.

Infection of both pleural sacs simultaneously is very rare. I have met with one such instance in which a bilateral croupous pneumonia was followed by a bilateral purulent effusion.

In considering the morbid anatomy of pleurisy it was pointed out that



G. 347.—Pyothorax (right-sided). 1, Esophagus. 2, Descending aorta. 3, Right e. 4, Right ventricle. 5, Left auricle. 6, Left ventricle.
e heart is displaced to the left and rotated on its long axis. The left lung is pushed ard and no longer overlaps the heart. The esophagus is also displaced to the left.

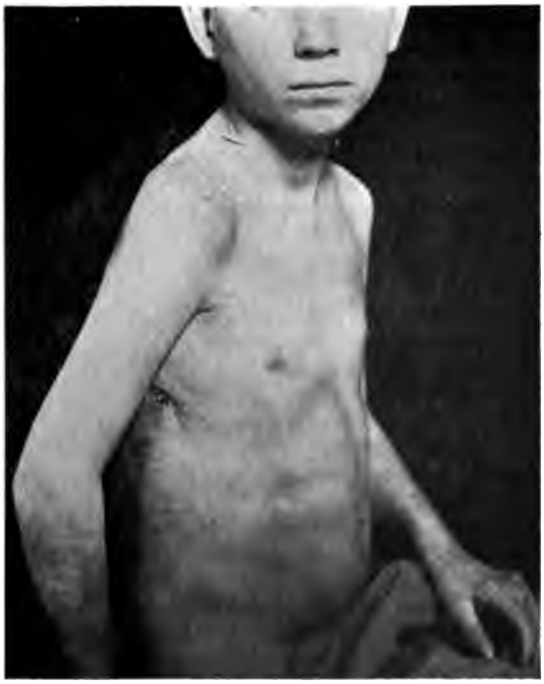


FIG. 348.—Empyema necessitatis.

the lung, as the result of pressure, became partially or completely collapsed. If the pressure is maintained for too long a period, the atelectatic part undergoes fibroid changes. Prolonged compression of the lung by a purulent effusion is probably one of the commonest causes of unilateral fibrosis of the lung.

Pryor[1] has recently called attention to a phase of empyema to which little notice has been paid, namely, the crippling effect the disease often has on the *diaphragm*. When empyema develops diaphragmatis is apt to ensue. The diaphragm becomes swollen, thickened and stiff. As the result of inflammatory changes it loses its elasticity and becomes rigid; later it becomes atrophied, the tissues undergoing fibroid changes and becoming hardened (see p. 642). If the effusion is large the weight of the fluid may flatten the diaphragm or cause it to become concave in shape and, at times, produce a tumor below the costal margin.

Symptoms.—The onset of a purulent effusion may be abrupt as in the case of serous fluid. When empyema occurs as a complication of one of the acute infections, notably pneumonia, the exudation of the purulent fluid may be very rapid, and a very considerable amount may accumulate as early as the fourth or fifth day of the disease. Not frequently, however, it has an insidious onset and is unsuspected, either because it develops during the course of other diseases or supervenes upon a preëxisting serous effusion.

In the acute cases the symptoms are practically identical with acute pleurisy. If the onset of the empyema is gradual, respiratory symptoms may be wanting entirely. Cough may or may not be present and unless the effusion is very large there will be no dyspnea. Some loss of weight, malaise and anemia are usually present. Sweating and irregular fever are common, especially in children.

In the great majority of cases of empyema there is a well-marked polynuclear leukocytosis. This fact will often serve to distinguish between serous and purulent effusions, although it is to be borne in mind that there may be a low white count even in the presence of pus.

Physical Signs.—For the most part the physical signs which occur in cases of empyema are identical with those encountered in cases of serous effusion. There are in addition, however, several additional features which are worthy of note.

Inspection.—Except in the acute cases, an individual suffering from an empyema is apt to reveal the evidences of a chronic suppurative process, namely, fever, sweating, loss of vigor, always some emaciation, and anemia. In chronic empyema clubbing of the fingers is often a marked feature of the disease and, in common with bronchiectasis and congenital heart disease, furnishes the most extreme grades of the condition encountered. In cases of empyema the clubbing of the fingers may develop very rapidly and instances have been recorded in which the condition was apparent within two or three weeks. It has been noted also that the clubbing disappears with the removal of the pus from the pleural cavity.

Bulging of the affected side and of the interspaces is more frequently encountered in cases of purulent than in the case of serous effusions, due, in all probability, to the greater weight of the fluid. In children bulging of the affected side may be very distinct. Tenderness on pressure is commonly present. For some reason displacement of the heart is apt to be more marked. If the empyema has existed for some time, the small ıtaneous veins on the affected side may be very noticeable. Edema e chest wall is frequently met with. In a certain proportion of of pulsation of the chest wall is noted over the area overlying the ema (see Figs. 105 and 107).

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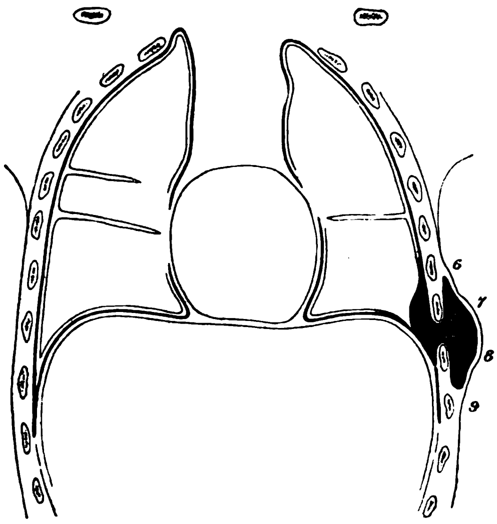
[1] *International Clinics*, June, 1916.

ulsating empyemata, while not invariably so, are in the vast majority es left-sided. Levi[1] found that the left side was the seat of a pulsat- fusion in 93.6 per cent. of 110 cases. In a few instances pulsation is in cases of serous effusion. Honeij[2] has reported a case in which pulsation was noted over a left-sided hydropneumothorax. The tion occurs more frequently in men than in women. In about alf of the cases the effusion occurs as a complication of pulmonary culosis; next to tuberculosis pneumonia is the most frequent exciting . Although the pulsation is synchronous with the cardiac systole ıtisfactory explanation has been forthcoming as to how the heart lse is transmitted through the effusion.



49.—Encapsulated empyema (necessitatis) discharging into subcutaneous tissues reen seventh and eighth ribs. (*Ashhurst, International Clinics,* vol. iv, 26th Series.)

ess frequently a localized tumor is seen on the chest wall caused by ıurrowing of the pus through the thoracic parietes (Figs. 348 and This is known as *empyema necessitatis.* Complete perforation of hest wall may result in the formation of a fistula. An empyema sitatis is often pulsating in character. Of 95 cases of pulsating ema analyzed by Sailer[3] there was in addition, an empyema neces- ı in 38. When an empyema necessitatis develops it almost invariappears on the anterior or lateral chest wall, somewhere between ırd and sixth interspaces.

lpation.—The presence of edema may be demonstrated by pres- ıith the finger tips when it would not otherwise be suspected.

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[1] *Centralblatt f. d. Grenzgebiete d. med. u. chir.,* xviii, No. 3, 1914.
[2] *Archives of Internal Medicine,* Oct., 1917.
[3] *Am. Jour. Med. Sc.,* cxxviii, 225, 1904.

Percussion.—In cases of empyema the liver dulness usually extends much farther below the margin of the ribs than in cases of serous effusion. This is due to the greater weight of the fluid which not only flattens the convexity of the upper surface of the diaphragm but may even cause it to become concave.

Auscultation.—The same variations in the character of the breath sounds occur in empyema as in serous effusions. The respiratory murmur may be faintly heard or entirely absent. Occasionally, and especially in children with a large effusion, the breathing may be loud and bronchial in character.

The transmission of the whispered voice through a serous effusion and the absence of such transmission in case pus is present, Baccelli's sign, has been alluded to. The sign is of no great value.

Diagnosis.—In a patient with the physical signs of a pleural effusion and in whom there are symptoms of sepsis, it is highly probable that an empyema is present. The use of an exploring needle will definitely decide the matter.

STREPTOCOCCUS EMPYEMA

During the winter of 1917–1918 there occurred in a number of the army cantonments a severe type of broncho-pneumonia which was frequently complicated by empyema. The mortality rate was exceedingly high. It was apparent from both the clinical picture and the bacteriological findings that these empyemas were caused by a virulent infection such as had but rarely been encountered in civil practice. Furthermore, experience in the recent epidemic of influenza has also brought to light a form of broncho-pneumonia (often complicated by empyema) which closely resembles the type of infection under discussion (see article on Influenza). The unusual features presented by this type of empyema seems to warrant a special description of its manifestations.

Etiology.—The organism which causes the trouble is a streptococcus and in the great majority of cases it is the streptococcus hemolyticus. When attention was first focused on this infection, it was noted that it followed an epidemic of *measles* and it was believed that there was a direct relationship between the two. This view was held by Cole and MacCallum in their study of the epidemic at Fort Sam Houston, Texas. Later, however, similar epidemics occurred in widely separated parts of the country in which measles played little or no part.

It now seems probable that infection with this organism occurs either as the result of exposure, or fatigue or, because an inflammation of the upper respiratory tract and bronchi, such as occurs in measles or influenza, renders these parts vulnerable. This assumption is strengthened by the fact that it has been demonstrated that many of the soldiers were carriers of the streptococcus hemolyticus, and in many instances, attacks of streptococcic tonsillitis or pharyngitis preceded the invasion of the lower respiratory tract.

Smears and cultures taken from the throat in 366 cases were shown by Blanton and Irons[1] to contain hemolyzing streptococci in 34 per cent.; non-hemolyzing streptococci in 33 per cent.; pneumococci in 12 per cent.;

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[1] *Jour. Amer. Med. Assoc.*, Dec. 14, 1918.

nd influenza bacilli in 8 per cent. Fox and Hamburger[1] state that
3 per cent. of one company of men carried hemolytic streptococci
ı the pharynx. Apparently the organism spreads from individual to
ıdividual with relative ease. Among measles patients admitted to
he hospital MacCallum[2] found a relatively small percentage of throats
ıfected with the hemolytic streptococcus, but they acquired this in-
ection rapidly when kept together in the wards and in many instances
eveloped a severe broncho-pneumonia. On the other hand, the fre-
uency with which the men were found to harbor hemolytic streptococci
ı their throats makes it highly probable that in some instances the in-
ection is an autogenous one and that the streptococcic infection is
uperimposed on the pneumonia rather than being the cause. Certainly
his sequence of events seemed to be true in many cases in the influenza
pidemic. In several of the camps it was noted that lobar pneumonia of a
ıild type and low mortality rate was succeeded by a severe type, as-
uming epidemic proportions, following the admission to these camps
f negro regiments.

In addition to being present in cultures from the throat the strepto-
occus hemolyticus has been cultivated from the sputum and especially
rom the pleural exudate. Miller and Lusk[3] report the finding of hemo-
ytic streptococci in 88 out of 95 exudates. It has not been recovered,
xcept in rare instances, from the circulating blood.

One of the characteristic features of these streptococcic broncho-
neumonias is the high incidence of empyema. Of 283 cases of pneu-
nonia in which careful bacteriological examinations of the sputum were
nade Brooks and Cecil[4] found that 136, or almost half, were strepto-
occic in origin. There were 49 cases of streptococcus empyema, show-
ng an incidence of empyema of 36 per cent. Contrasted with this group
s an equal number of pneumococcus-pneumonias in which the incidence
f empyema was but 11 per cent. In addition there were 15 cases of
terile empyema which were about equally divided between strepto-
occus and pneumococcus pneumonia. In the epidemic studied by
Miller and Lusk[5] the incidence of empyema was 34.8 per cent.

Morbid Anatomy.—This type of empyema, although commonly asso-
iated with broncho-pneumonia, may occur with very little or even no
emonstrable pulmonary change. In such instances infection of the
leura may be by way of the lymphatics or by extension from a small
bscess beneath the visceral pleura. Several of the reports seemed to
ıdicate that the empyema was most apt to occur in those cases most
verely infected. It develops with surprising rapidity and often before
ıe pneumonia is definitely recognized. It is then seen that these
npyemas are imposed on, and do not merely succeed, the active stages
pneumonia. In this particular they differ from pneumococcic empy-
ıas which usually occur late in the course of the disease when the
tient is convalescent (Brooks and Cecil).

The associated pulmonary lesion is usually an interstitial broncho-
eumonia but other forms characterized either by small miliary areas,
embling tubercles, or large confluent areas may occur. The pleura

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[1] *Jour. Amer. Med. Assoc.*, June 8, 1918.

[2] *Ibid.*, Aug. 31, 1918.

[3] *Loc. cit.*

[4] *Archives of Internal Medicine*, Sept., 1918.

[5] *Jour. Amer. Med. Assoc.*, Aug. 31, 1918.

on the affected side is covered with a fibrinous coat, varying in thickness, and closely adherent. This fibrinous coating may be converted into a thick coat of granulation tissue over the surface of which is a layer of fibrin and leukocytes.

The fluid may be sacculated at one or more points or be free in the pleural cavity. The fluid, if removed early, is usually of a light yellow color, with, at times a greenish tinge and may be clear or only slightly cloudy. In several cases I have seen the fluid was slightly hemorrhagic in character. When removed later in the disease the fluid is usually distinctly purulent. The bacterial content of the fluid in these empyemas is notably higher than that encountered in the pneumonic type, plate cultures from a single loop of the culture, sometimes showing from 100,000 to 200,000 colonies.

Of associated lesions the most frequent is a *suppurative pericarditis*, no less than eight examples of this complication occurring in thirty-six cases of streptococcic empyema observed by Brooks and Cecil. The involvement of this serous membrane may occur by direct contiguity or by way of the lymphatics. *Abscess of the lungs* is not infrequent. *Ulcerative laryngitis*, which may cause destructive changes in the vocal cords and epiglottis was noted by MacCallum in the more acute cases and especially in those following measles.

The mortality rate is exceedingly high, being over 50 per cent. in some localities.

Symptoms.—In many instances the patients were suddenly prostrated and showed the evidences of a severe intoxication. In others, the onset was gradual and there was a history of an attack of pharyngitis, bronchitis, or pain in the side. The cough is often dry and unproductive at the onset. The sputum is yellowish in color and viscid. The rusty or hemorrhagic sputum so common in lobar pneumonia is rarely present. Profuse sweating which involves the entire surface of the body is very suggestive of a developing empyema. The sweating may be more marked about or even confined to the head and neck. A chill is not often noted in association with the sweating.

The fever is irregular, the pulse rate proportionately slow and the respirations, as a rule, high. Acceleration of the respiratory rate often occurs when an empyema develops and in not a few of the cases the exhaustion becomes more marked. Pain is not constantly present and when it is, is rarely of long duration.

The white cell count is not of much value in determining whether there is a complicating empyema or not.

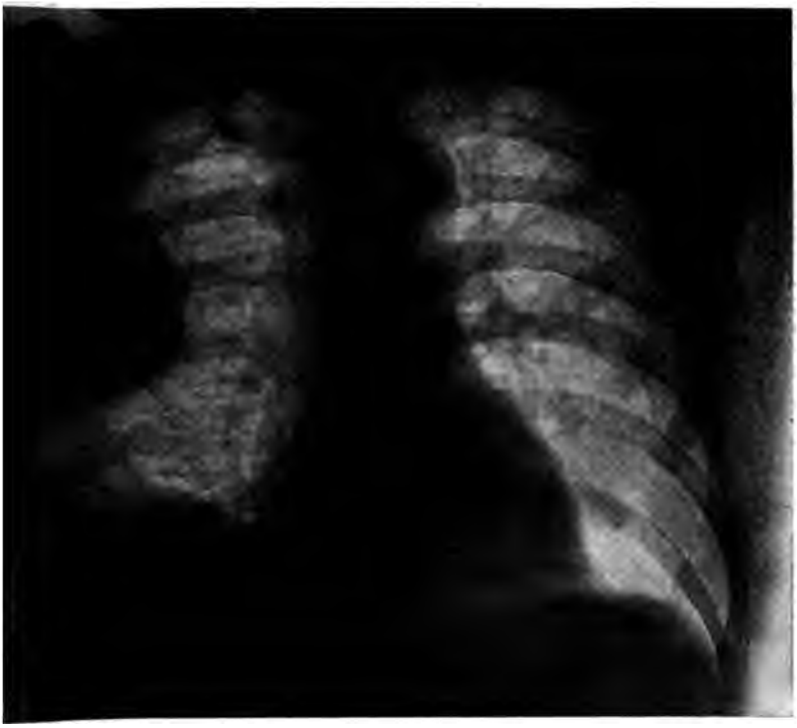
The empyema may develop very early in the attack but in the majority of cases it first becomes manifest on the fourth or fifth day of the disease.

Physical Signs.—All of the reports indicate that the physical signs were often inconclusive. On *inspection* the most important thing is the location of the apex beat of the heart. The displacement of the apex beat is unquestionably one of the most important signs we have and in doubtful cases may be the decisive factor.

On *palpation* restriction of the affected side may be noted. A very suggestive finding is an area of localized tenderness on deep pressure.

On *percussion* the note over the fluid is flat and high pitched. Tenderness over the affected area is frequently elicited on percussion.

signs obtained on *auscultation* are often confusing. In this type of na and in many of those complicating influenza, the transmission bronchial breathing and both the spoken and whispered voice the fluid is exceedingly common. In this respect they differ lly from the great majority of cases of serous effusions and empye- to other causes. Why, in some instances, the breath and voice re transmitted through an effusion and in others are not, has been ely explained by Montgomery and Eckhardt (see pages 60 and 61). en voice in these cases often has a distinct nasal quality and when is suggestive of fluid rather than consolidation. Inasmuch as



350.—Encysted empyema between upper and middle lobes. At operation the wall was tough and almost cartilaginous. About 2 ounces of creamy, fetid pus oved. (*Dr. D. R. Bowen.*)

ement of the outer layer of the pericardium is often present the ence of a pleuropericardial friction rub is not uncommon; it ears later as the effusion develops. As pericarditis has been found a relatively frequent complication the precordium should be care- usculted for the presence of a friction rub.

a few instances the signs of a pneumothorax are added to the e. This condition is transient and the cause unknown.

agnosis.—In a patient suffering from a streptococcic broncho- onia a complicating empyema is to be suspected if there are signs reasing toxemia, profuse sweating, acceleration of the respiratory nd the occurrence of pain or localized tenderness on pressure or from moderately forcible percussion. The development of bronchia breathing and egophony at the base of the chest in this type of pneumonia is always suggestive of an effusion. In suspected cases the exploring needle should be freely used and whenever possible, an X-ray examination should be made.

LOCULATED OR ENCAPSULATED EMPYEMA

An encapsulated collection of fluid, almost always purulent, but sometimes serous in character, may occur in one of the fissures between the lobes of the lung, between the diaphragm and under surface of the base of the lung, between the lung and pericardium, or as the result of adhesions forming pockets between the visceral and parietal layers of the pleura. The latter may occur at any point within the pleural sac but are found most commonly in the lower and posterior aspects of the chest (see Figs. 350, 351, 352, 353).

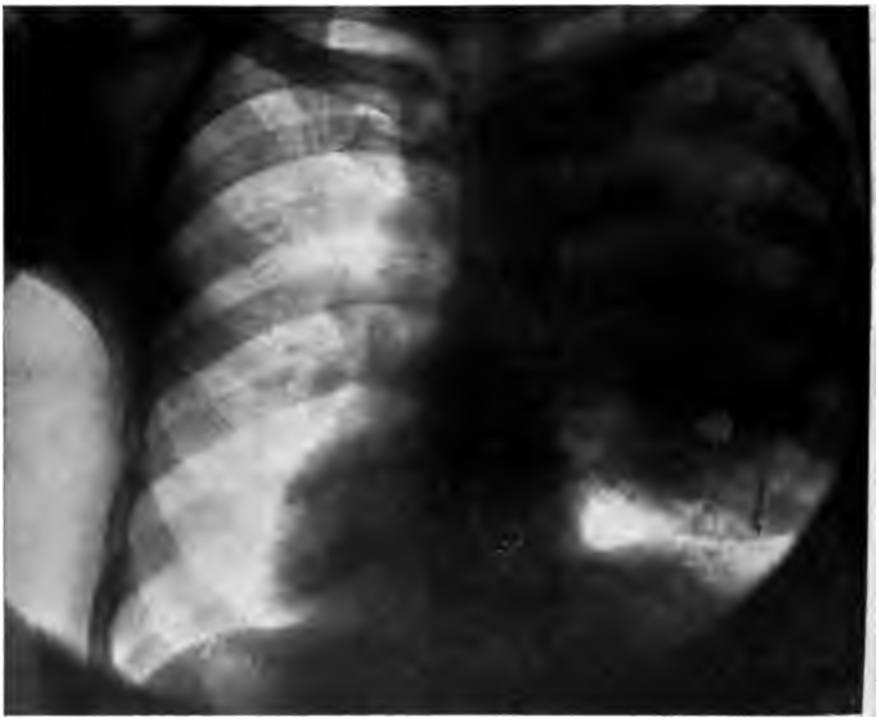
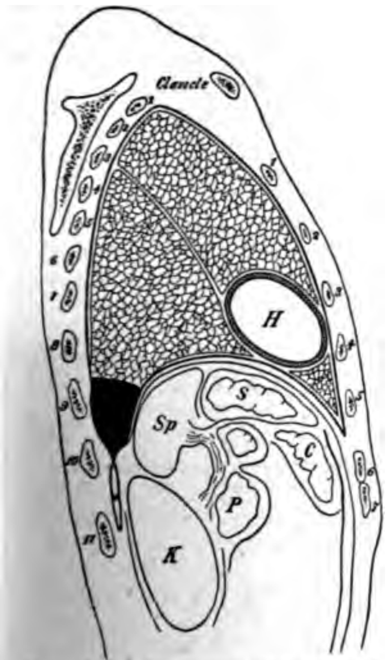


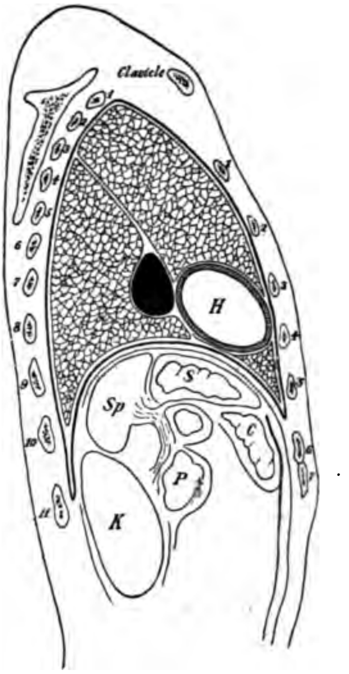
Fig. 351.—Localized apical empyema. (*Dr. D. R. Bowen.*)

Inasmuch as a collection of pus in one of the interlobar fissures is not uncommon, it is important to keep in mind the position of these fissures. Figs. 354 and 355 show their location. Fig. 355 shows the complementary pleural space posteriorly.

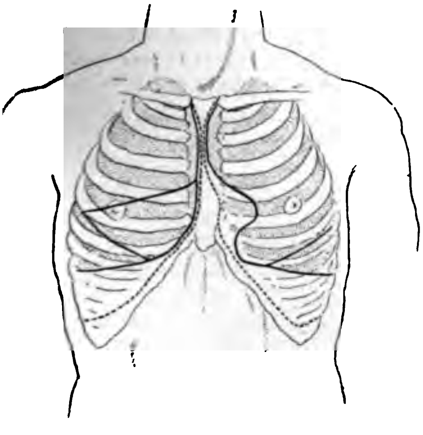
Etiology.—Fluid may become encysted in any one of the sites above mentioned under the same conditions under which free pus in the pleural



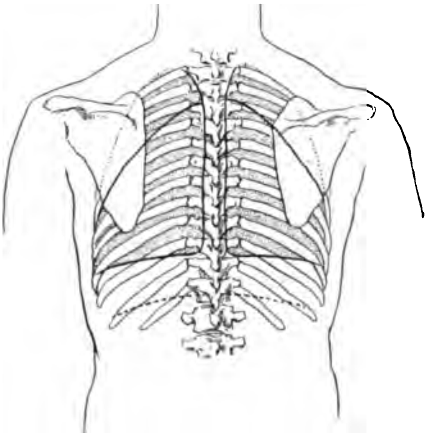
Fɪɢ. 352.—Empyema encapsulated at base of left lung. Sagittal section of thorax through middle of left clavicle. (*Ashhurst, International Clinics*, vol. iv, 26th Series.)



Fɪɢ. 353.—Diagram of sagittal section through middle of left clavicle, indicating site at which pus was found at autopsy. (*Ashhurst, International Clinics*, vol. iv, 26th Series.)



Fɪɢ. 354.—Anterior view of fissures of lungs Dotted lines indicate the pleura.



Fɪɢ. 355.—Posterior view of fissures of lungs. Dotted lines indicate lower limit of pleural sacs.

sac or a pulmonary abscess occurs. The condition is considered as being relatively rare. Among 35,900 admissions to the City Hospital, of St. Louis, Elsworth Smith[1] reports 87 cases of empyema and of this number 3 or 3.4 per cent., were of the encysted type. Lord[2] found practically the same percentage, 3.2, in 248 cases of empyema. On the other hand, James[3] of Edinburgh, has reported 19 instances of encapsulated fluid among 43 cases of empyema. By most observers the term encapsulated empyema is restricted to small collections of pus located in the interlobar fissures or between the lung and the diaphragm while relatively large purulent effusions which may occupy as much as half the pleural cavity are classed as ordinary empyemas. Ashhurst[4] very properly points out that with the exception of massive empyemas, the pus is always encapsulated (see Figs. 346 and 347). Adhesions are quickly formed between the lung and adjacent structures and in this way the pus is walled off. In some instances the resulting vomica is small, in others it may occupy a large portion of the pleural cavity. In rare instances the encysted fluid is serofibrinous in character. Pleural effusions which arise as the result of a transudate (heart and renal disease) never become encapsulated.

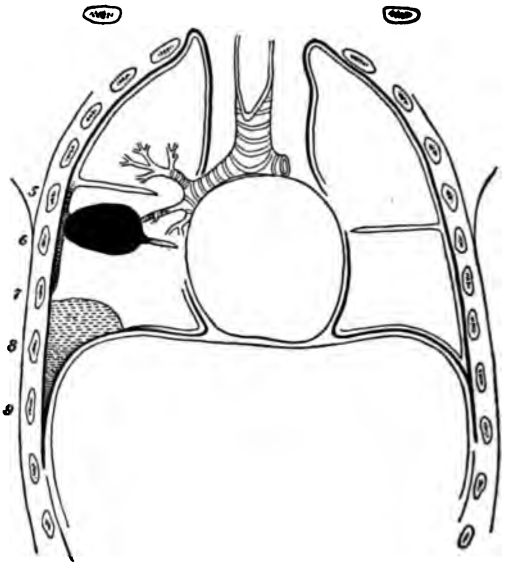


Fig. 356.—Interlobar empyema ruptured into a bronchus (pleural vomica). Note pleural effusion in costo-phrenic sinus. (*Ashhurst, International Clinics*, vol. iv, 26th Series.)

Morbid Anatomy.—As the result of a fibrinous exudate adhesions may take place at the circumference of the inflammatory area thus walling off the fluid if any arises. This may occur between the under surface of the lung and the diaphragm or at any point over the antero- or postero-lateral aspects of the lung. In like manner the exudate may extend slightly into one of the fissures dividing the lobes. In the latter instance if adhesions occur a closed pocket is produced.

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[1] *Jour. Am. Med. Assoc.*, Dec. 7, 1912.
[2] "Diseases of the Bronchi, Lungs and Pleura," p. 531, 1915.
[3] "Pleurisy," p. 139.
[4] *International Clinics*, vol. iv, twenty-sixth series.

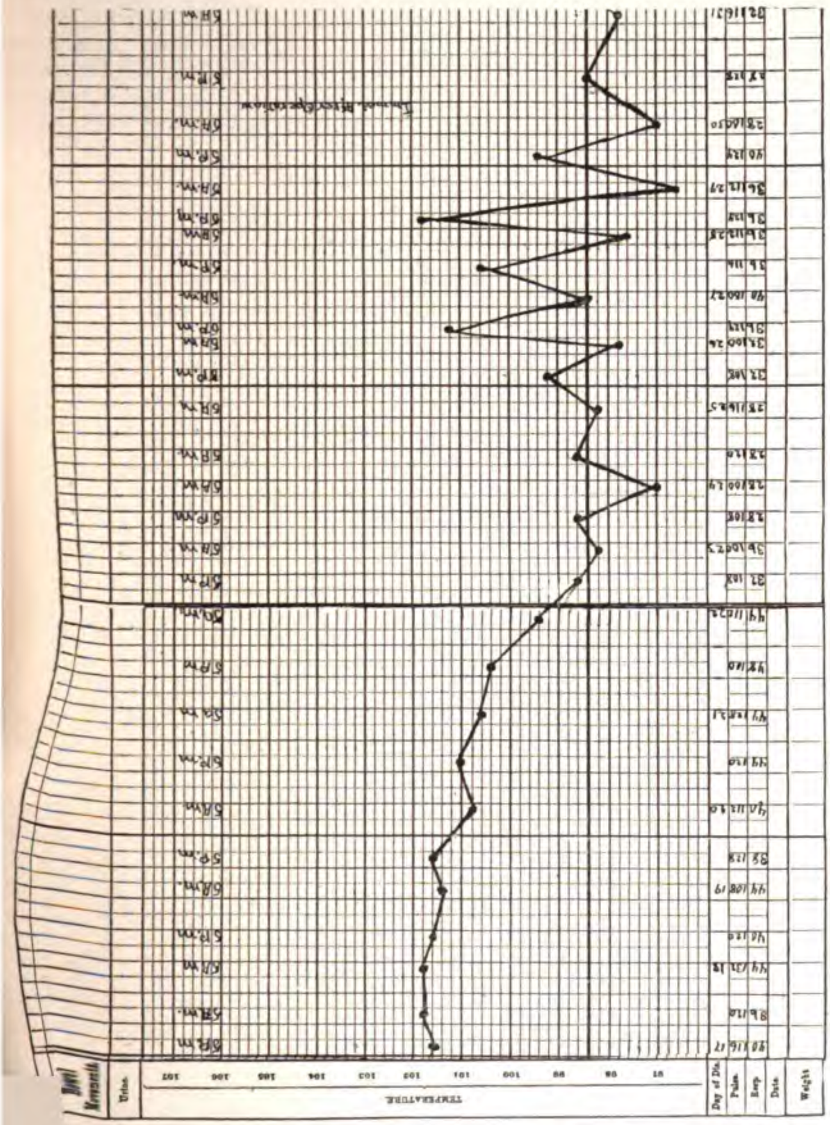

FIG. 357.—Loculated empyema.

many instances what is believed to be a pulmonary abscess is in
y an encapsulated empyema which has ruptured into a bronchus
356). On the other hand, a pulmonary abscess may perforate the
ral layer of the pleura. The pus may thus become encysted and
an encapsulated empyema.

The amount of pus is variable, ranging from 1 or 2 ounces to as much as 2 pints.

Symptoms.—An empyema which is encysted may declare itself in much the same manner as the ordinary empyema. The patient may be seized with sudden pain in the side followed a few days later by cough and expectoration. Fever of the type usually seen in septic cases is usually present (Fig. 357). This is especially true if the pus is located between the lung and diaphragm or between the visceral and parietal layers of the pleura. If the collection of pus is considerable in amount the constitutional symptoms are apt to be marked.

If, however, the pus develops between the lobar fissures and is small in amount, the onset may be frank with pain and fever or it may be insidious and the symptoms indefinite. In such cases the picture presented is suggestive of a small focus of suppuration, namely, fever, with marked remissions or intermissions, anorexia, and malaise. Pus located between the fissures acts as a foreign body and thus produces cough and expectoration. As already stated the first intimation of the trouble may be the expectoration of a considerable amount of pus as the result of rupture of the pus into a bronchus.

Physical Signs.—*Inspection.*—If the amount of pus is small and has been present only for a short time, nothing of note will be seen on inspection. If, on the other hand, it has been present for some time it will produce much the same effect as a foreign body. Thus the surrounding lung tissue may be congested, atelectatic or even fibroid in character. In such cases there will be more or less restriction of expansion on the affected side and in addition the chest wall may be retracted.

Palpation.—If one has reason to suspect that an encysted empyema may be present the chest should be palpated by means of firm pressure with the finger tips. This may reveal a localized area of edema or a sharply circumscribed area of tenderness. The latter is very suggestive of the presence of localized pus. Tactile fremitus is variable; its presence or absence will depend largely on the size of the effusion.

Percussion.—This should be performed lightly in order to distinguish between the flatness over the fluid and the dulness over the compressed lung tissue surrounding the fluid.

Auscultation.—If the effusion is sufficiently large, there may be a slight area over the fluid and about this broncho-vesicular or bronchial breathing and mixed râles over the collapsed or congested lung. In the same manner the voice sounds may be absent at one point and slightly increased in others.

Diagnosis.—When all is said and done the recognition of an encysted empyema is extremely difficult. The history, the symptoms and the physical signs are rarely conclusive; as a rule, they are only suggestive.

Inasmuch as an encysted empyema is not an infrequent sequel of *lobar pneumonia* the persistence of symptoms, leucocytosis and physical signs after the termination of an attack of pneumonia should arouse suspicion as to the presence of loculated pus. I have emphasized elsewhere the fact that delayed resolution is an infrequent termination of pneumonia. In the great majority of cases believed to be due to delayed resolution, the actual cause of the failure of the lung to return to its normal state, is the presence of pus in the pleural cavity. The pus may be free or encysted.

The development of pulmonary symptoms a week or so after a surgical operation also should arouse the suspicion that pus may be present in the chest. This is especially true in suppurative conditions involving the abdominal viscera.

In common with other long-standing pulmonary infections a loculated empyema is very frequently mistaken for *tuberculosis*. The cough, expectoration, slight fever, and malaise are very suggestive but the absence of tubercle bacilli from the sputum and the location of the abnormal physical signs should serve to eliminate tuberculosis.

The exploring needle should be used if the evidence points strongly to the presence of fluid. In addition every case with respiratory symptoms the cause of which is obscure, should be examined by means of the X-rays. It is to be borne in mind, however, that neither needling nor the X-ray is infallible and this is especially true of the former. The needle may not be introduced in the right direction or the pus may be too thick to be withdrawn.

Exploratory thoracotomy has been so perfected that the operation can be performed with as little risk as an abdominal exploration. It should be recommended, therefore, in all cases whether the pus is definitely located or not.

HEMORRHAGIC PLEURAL EFFUSIONS

As the majority of pleural effusions are inflammatory in origin it is no unusual occurrence to find red blood cells in the fluid. In the great majority of instances the number of red corpuscles is too small to cause any alteration in the color of the effusion. Dieulafoy as the result of histological studies of pleural fluids, found that the presence of from 1500 to 3000 red cells per cubic millimeter caused no appreciable alteration in the color of the fluid. If, however, the number of red corpuscles reaches 5000 or 6000 per cubic millimeter a rosy tint is imparted to the fluid and the higher the cell content the more markedly hemorrhagic does the effusion become. A curious feature of hemorrhagic effusions is the occasional presence of an excessive number of eosinophiles not only in the fluid itself but also in the circulating blood. The same thing has been noted in cases of hemothorax. Klein[1] has recorded a case in which the pleural fluid showed 76.4 per cent. and the systemic blood 40 per cent. eosinophiles; Dieulafoy's case had 35 per cent. in the effusion and 10 per cent. in the circulating blood. In other instances an excess of eosinophiles may be present in the pleural fluid and not in the circulating blood and *vice versa*. Thus Harmsen[2] found 8.64 per cent. of eosinophiles in the pleural fluid alone; on the other hand, Lord observed a case in which there were no eosinophiles in the effusion but 20 per cent. of the white cells in the circulating blood were of that character.

The discovery of a hemorrhagic effusion is almost invariably an accidental finding. Occasionally its presence may be suspected when an effusion develops in an individual known to be cancerous. As effusions which develop secondarily to malignant disease of the mediastinum, lungs or pleura are quite as often serofibrinous as hemorrhagic and the latter have no distinctive signs, it can be seen that such a diagnosis prior to racentesis is nothing more than a guess. A hemorrhagic effusion is encountered in the following conditions: 1. *Tuberculosis.* This is probably the most common cause of blood-stained effusions. As has been pointed out in the section on pleurisy the first manifestation of tuberculosis may be a pleural effusion. Rarely the effusion is hemorrhagic in character. It is the result of a primary tuberculosis of the pleura or the coincident involvement of the lung and overlying pleura. The hemorrhagic character of the fluid is to be explained by the congestion which surrounds the newly formed tubercles and the vascularity of the new tissue. Leakage occurs later partly as the result of degeneration of the small vessels and partly as the result of involvement of the vessels of the tuberculous process.

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[1] *Cent. f. innere Med.*, Jan. 28, 1899.

[2] Quoted by Klein.

The condition is analogous to an hemoptysis which may be the immediate forerunner of active tuberculosis or there may be an indefinite period of latency. As an early manifestation the effusion is apt to occur in individuals who are apparently healthy. An excess of lymphocytes in the fluid is very suggestive of tuberculosis. In any case the sediment from the fluid should be examined for the presence of tubercle bacilli and in their absence animal inoculations should be made.

More rarely an acute miliary tuberculosis or a rapidly advancing tuberculous broncho-pneumonia is associated with an effusion which is hemorrhagic in character. This is also to be ascribed to involvement of the pleura.

Occasionally blood-stained effusions are encountered in the ordinary chronic type of tuberculosis. The reaccumulation of the fluid is very common. It must be borne in mind, however, that the same thing is commonly seen in malignant disease and occasionally in hemorrhagic effusions of an apparently benign origin. Hemorrhagic effusions, tuberculous in origin, may, after several tappings, become serous in character.

2. *Malignant Disease.*—This is a well-recognized cause of hemorrhagic effusions. In individuals who are known to be suffering from malignant disease in some portion of the body or who have been operated on some time previously for a malignant tumor, the occurrence of a hemorrhagic effusion is the strongest kind of evidence that metastasis to the thoracic viscera has taken place. Such cases offer little difficulty. If, however, the effusion is secondary to primary malignant disease in the mediastinum, lungs or pleura which, as yet, has given no evidence of its presence, the diagnosis may remain doubtful for some time. Ross[1] in 60 cases of malignant disease involving the mediastinum found an effusion in 29, and of this number 15 were hemorrhagic. Rapid reaccumulation of the fluid is suggestive of both tuberculosis and malignant disease and is apt to be more persistent in the latter. Dieulafoy records a case in which thoracentesis was performed thirty-three times in four months, and 44 pints of hemorrhagic fluid withdrawn.

Cytological examination of the fluid will show the absence of lymphocytes and tubercle bacilli. The finding of large masses of cells or the so-called cancer cells is looked upon by some observers as strong evidence of the presence of malignant disease.

3. *Asthenic Conditions.*—Occasionally a hemorrhagic effusion is encountered in association with conditions characterized by marked asthenia. Among these may be mentioned the malignant types of the

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[1] *Edinburgh Med. Jour.,* December, 1914.

acute infectious fevers, scurvy and occasionally, the transudates occurring in the course of chronic nephritis and cardiac disease. Pressure on the azygos veins is also given as a rare cause of a hemorrhagic transudate. Hemorrhagic effusions are not uncommon in association with cirrhosis of the liver but in the majority of these cases tuberculosis is also present and is to be looked upon as the actual cause.

4. *Simple or Idiopathic Cases.*—Not infrequently a hemorrhagic effusion occurs in an individual in whom there is no evidence of either tuberculosis or malignant disease. Osler refers to an instance in which the patient was entirely well eight years later and Cheesman and Ely[1] have recorded a case in which the pleural effusion reaccumulated for a period of eighteen months and the peritoneal fluid for nearly five years. This patient also showed no evidence of either tuberculosis or malignant disease. These are exceptional cases. In the majority of instances these idiopathic cases are to be looked upon as tuberculous although the interval between the appearance of the hemorrhagic effusion and definite tuberculosis may be a long one. The same may be said of *hematoma of the pleura* which is given as a rare cause of hemorrhagic effusion.

5. Finally mention should be made of the fact that in performing the operation of thoracentesis a small vessel may be punctured and blood in this way may become mixed with a serofibrinous exudate. A rare accident is puncture of an intercostal artery with the aspirating needle. Death resulted from this accident in a case observed by Horder.[2]

The symptoms and physical signs are the same as those occurring in serous effusions.

HEMOTHORAX

Etiology.—An effusion of blood into the pleural cavity may occur as the result of rupture of an intrathoracic blood-vessel or of an intercostal artery. The latter is by far the most common. There are two groups of cases: the non-traumatic and the traumatic.

1. The NON-TRAUMATIC cases are due to rupture of a blood-vessel which is, as a rule, diseased. The most common cause is aneurism. Occasionally the rupture of a thoracic aneurism may lead to the escape of an enormous quantity of blood into the pleural cavity. When rupture into a pleural cavity does occur, it is usually on the left side. Among 22 cases collected by Goodman,[3] rupture into the left pleural cavity occurred in 7, while the right side was involved but once. Fowler observed a case in which a fatal issue occurred as the result of rupture of a small aneurism of one of the internal mammary arteries. Caries of one of the ribs may cause erosion of an intercostal artery followed by hemorrhage into the pleural cavity.

Tumors by causing pressure on the larger intrathoracic veins, may lead to the establishment of a collateral circulation. The smaller veins which take up the burden of the circulation may become greatly dilated varicose and one of these varicosities may rupture (West).

Erosion of one of the intrathoracic vessels by cancer, or an abscess, n occasional cause of hemorrhage. Rarely an effusion of pure blood the pleural cavity occurs in cases of scurvy and hemophilia.

Jour. Med. Sc., August, 1899.

tt and Rolleston: "System of Medicine," vol. v, p. 559.

. *Med. Assoc.*, April 18, 1914.

2. TRAUMATIC HEMOTHORAX.—This is by far the most common variety. Penetrating wounds of the chest are not especially common in civil life but in military practice they are of frequent occurrence and a very high proportion of them are followed by a hemothorax. It is important to bear in mind that the bleeding which follows a penetrating wound of the chest does not come from the perforated lung but from the parietal pleura. "When we consider how readily the intercostal vessels may be torn by a bullet or by a fragment of fractured rib projected by the bullet, this is not surprising" (Keogh Murphy). It is, of course, understood that this applies to wounds beyond the middle line. Perforation of the large vessels at the root of the lung or a great vein at the root of the neck may be followed by a rapid and fatal hemorrhage into the pleural cavity.

Penetrating wounds may be produced in the following ways: (1) A high velocity bullet may perforate the chest, causing only minute wounds, of entrance and exit, associated with but little bleeding, pneumothorax or pulmonary collapse. (2) A shell fragment may be lodged in the lung and cause profuse bleeding into the pleural cavity. In addition there are: (*a*) free air, free blood and a collapsed lung; or (*b*) there may be little or no free air, a large hemothorax and a compressed lung, the latter being Nature's usual method of checking hemorrhage. (3) The superficial wound may be large with extensive communication of the tubes and the wound of the (air) "sucking" variety. This type is usually associated with marked mediastinal displacement, dyspnea, shock, and ultimately if the patient survives, infection. As Makins[1] has pointed out, there is no reason why a perforation by a bullet of small caliber should be much more feared than a puncture from an exploring trocar. This is due to the fact that the lung is composed largely of elastic tissue which at once contracts closing up the hole made by the bullet; at the same time the contractile power effectively seals up the opening in the visceral pleura. Even relatively large vessels and bronchi may be perforated and sealed up by the elastic tissue surrounding them. Healing of the pleural surface takes place with great rapidity; so much so that, within three or four days of the injury, it will be difficult to find at post-mortem the actual track of the bullet through the lung (Murphy).

In Murphy's[2] experience penetrating wounds of the chest are complicated by a hemothorax in about 60 to 70 per cent. of all cases as the result of a gradual continued oozing from an intercostal vessel. The frequency varies enormously, however, with the early treatment. Prolonged rest immediately after the injury greatly reduces the incidence. If the wounded are transported some distance shortly after the injury is received the percentage of cases developing a hemothorax is very high. In the South African War, Makins pointed out that among the men who were at once brought to a base hospital and kept at rest, the hemothorax occurred in 30 per cent. but in those who were transported long distances, hemothorax occurred in no less than 90 per cent. of all penetrating wounds of the chest. Makins divides these cases into the following groups: (1) Gradual or primary hemothorax due to steady small oozing from the time the wound was received; (2) recurrent, where several hemorrhages have occurred; (3) secondary hemorrhage which is the same as that occurring elsewhere and apt to happen somewhere about the eighth or tenth day.

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[1] "Surgical Experiences in South Africa, 1899–1900."
[2] "Wounds of the Thorax in War," 1915.

The relative infrequency of penetrating wounds in civil life and the excellent hospital facilities in most communities explain to a great extent the rarity of hemothorax in any but military practice.

Symptoms.—The initial symptoms vary with the nature of the wound and the amount of bleeding. The rupture of a thoracic aneurism, or opening of a large vessel as the result of trauma, is followed by profound shock and death within a few minutes. If the source of the bleeding is from a torn intercostal artery the early symptoms may be so slight as to pass unnoticed. Usually, however, there is some evidence of shock, especially if the injury occurs in the lower part of the chest. The shock is to be attributed to the injury of the chest wall and the thoracic concussion rather than to that of the lung itself. The patient is pale, has a rapid and weak pulse and he may faint. External bleeding is usually very slight in amount. Pain is not a marked feature, especially after bullet wounds. There may be some pain and tenderness, however, at the point of entrance and exit, especially if a rib has been fractured. For a short time after receiving the wound the patient feels as though his "wind had been knocked out." There is some difficulty in getting the breath and a feeling of oppression in the chest. This, however, passes off. Hiccough and vomiting point strongly to perforation of the diaphragm.

Hemoptysis after a bullet wound is not uncommon. Blood-streaked sputum may be present for three or four days and then suddenly cease. Eloesser[1] states that blood-streaked sputum is not always an evidence of perforation of the lung as it may follow a severe contusion of the chest wall. If the bullet does not pierce a large vessel or injure an intercostal artery, little or no inconvenience is felt. There is slight tenderness at the point of entrance and exit, difficulty in breathing and a sense of oppression immediately after the injury and possibly some blood-streaked sputum for a few days. At the end of this time the individual is again ready for duty.

If the hemorrhage into the pleural cavity has occurred, there is apt to be some fever about the third or fourth day. The temperature is irregular and if recurrent bleeding takes place the temperature again rises. Secondary rises in the temperature are not always due, however, to fresh bleeding; they may be caused by infection, mention of which will be made later.

If the hemorrhage is severe but not immediately fatal, the skin is blanched and clammy, the pulse rate rapid and the temperature is raised and irregular in character. Murphy states that unless one of the great vessels is pierced, the hemorrhage following chest wounds is rarely fatal as the pressure of the distended pleural cavity will check the bleeding.

When blood has been poured out into the pleural cavity, it, after a
separates into two parts: (*a*) a thick, fibrinous, corpuscle enmesh-
ortion which accumulates posteriorly, especially at the base; takes
g time to absorb; forms adhesions; and is accountable for the per-
nt percussion dulness which lasts long past the usual convalescent
iod; and (*b*) a thin, dark red, or brownish red, or a "porter-like" fluid,
ich collects anteriorly and laterally, and constitutes the "blood"

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[1] *Jour. Am. Med. Assoc.*, Dec. 4, 1915.

which is withdrawn when aspiration is performed. If aspiration is delayed for three or four weeks sedimentation may take place and the fluid withdrawn is similar to serum. Brick-red colored, grumous fluid is characteristic of infection (Makins).

After gunshot wounds, infection is of frequent occurrence and unless recognized death is apt to ensue. In Bradford's[1] experience approximately 25 per cent. of the cases of hemothorax were infected. In 80 per cent. of these cases the infection was carried in from the skin or clothing; in the remaining 20 per cent. the infection was caused by organisms derived from the lung. In the early days of the European war the symptoms were believed to be due to secondary hemorrhage but post-mortem examinations showed that infection of the hemothorax had occurred.

Small or moderately sized hemorrhages into the pleural cavity are not necessarily serious. Their chief danger lies in their becoming infected. If the infection can be avoided or detected early the great majority of the cases recover. In Norris' experience a persistent or increasing fever or a rapid and rising pulse rate are the most reliable indices of an infected hemothorax. Constitutional signs such as anorexia, restlessness, increasing pallor, and insomnia are important signs but usually rather late manifestations. A sudden increase in tympany, cardiac displacement and the appearance of (often deep) jaundice point strongly toward gas-bacillus infection and indicate immediate resection and drainage. A fetid odor of the aspirated blood also points most emphatically to surgical intervention but one should be cautious against being misled by the stale, mawkish odor which old blood often possesses even when it is culturally quite sterile and innocuous.

In dealing with these cases Norris points out that one is often confronted with confusing facts. Thus a patient may show steady improvement, absence of constitutional symptoms, and a practically normal or constantly declining pulse rate and temperature, while the laboratory reports organisms microscopically or growth on culture media. In such cases it is probable that the infected area is walled off and that recovery may take place without a thoracotomy. Such cases should be carefully watched and at the first intimation of constitutional symptoms surgical intervention should be invoked. As a rule, the presence of the gas bacillus is an indication for surgical intervention no matter whether symptoms are present or absent. In contrast to other cases are those in which the bacteriological findings are negative and yet the patient has symptoms. In such cases a thoracotomy had best be done. Norris is of the opinion that one's judgment as to the course to be pursued is dependent to a great extent on the confidence one has in the bacteriologist.

Physical Signs.—*Inspection.*—When hemorrhage has taken place into the pleural cavity the affected side shows more or less bulging at the base and motion is much restricted. The apex beat of the heart may be displaced if the effusion is sufficiently large. If the effusion has become infected with a gas-producing organism, the heart is rapidly displaced.

Palpation.—Except as an aid in locating the position of the heart, palpation has usually less value than the other classical methods of examination. As a rule fremitus is diminished over the effusion and increased over the unaffected side. If the lung behind the effusion is arkedly compressed, fremitus will be present, as is the case in the patho- gic effusion of civil practice.

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[1] *Lancet*, Jan. 29, 1916.

Percussion.—A tympanitic note may occur under the following con- itions: (*a*) free air, (*b*) relaxed lung, (*c*) high position of the diaphragm,) gas bacillus infection. Tympany due to these causes is usually quite eadily differentiated from the hyperresonance encountered over the ound, previously functionating, side. Quite often, especially if the atient has been lying on his back, the whole anterior and lateral aspects f the chest may be tympanitic and only posteriorly will dulness be lemonstrable. It is often quite impossible to outline the cardiac dulness n one or the other side owing to the presence of such tympany. Not nfrequently Grocco's area of triangular dulness can be demonstrated, especially in large hemothoraces. Frequently this will be associated with bronchial breathing on the uninjured side, a physical sign which may result from mediastinal displacement either into or irritant compression of the sound lung. Such bronchial breathing may be mistaken for pneumonic consolidation. Movable dulness is sometimes demonstrated if free air is present but, as a rule, the blood is too clotted or the pleural cavity too full to permit of much, or of a rapid gravitation of blood. Nor is the elicitation of movable dulness worth the trouble its demonstration entails.

Auscultation.—Over the sound side the breath sounds are harsh, exaggerated and invariably associated with râles. The sounds are often so loud that they are transmitted to the injured side where, especially below the clavicle, they may give the impression that the upper lobe is functioning whereas this lung may be atelectatic or completely compressed. When in doubt in this regard, the degree of motion of the injured side, and the amount of cardiac displacement will often make the situation clear. Bronchial breathing, whether heard over the sound or the injured side, will nearly always indicate compression, and but rarely pneumonic consolidation. Amphoric or cavernous breath sounds, generally associated with whispered pectoriloquy point to hemo-pneumothorax and are often heard over chests with sucking sounds or after drainage tubes have been introduced.

Friction sounds are generally present and result from the sticky fibrinous moiety of the outpoured intra-pleural blood (see "Pericardial Knock," p. 257). The testing of vocal resonance by means of whispering is preferable in as much as it is less taxing to the patient.

Exploration.—A good sized exploratory needle and a reliable syringe should be unhesitatingly, freely and repeatedly used, not only for the purpose of demonstrating a hemothorax, which if small, is of minor mportance, but of determining the presence of infection, especially nfection by a gas-producing organism.

It is usually advisable on the third or fourth day after the injury to emove as much of the outpoured blood as possible. Secondary hemor- hage is usually small in amount, and the removal of large effusions arkedly shortens the period of convalescence, and gives great mechan- al relief to the respiratory and cardio-vascular functions.

X-ray Examination.—An X-ray examination is essential to determine e presence, size, shape and location of the missile. It is also useful in owing mediastinal displacement, pulmonary collapse, free air or blood in the pleural cavity. At times pus can be located which would otherwise be sought for in vain.

Abdominal Signs and Symptoms.—Injuries of the pleura, especially when in the neighborhood of the diaphragm often give rise not only to abdominal pain and rigidity but also to nausea and vomiting. These facts are important for the obvious reason that one frequently has to decide whether the missile which entered the pleura has penetrated the diaphragm, perforated a viscus or set up a peritonitis. Needless anesthetization, especially with ether, of a man who already has a wounded lung is of course most reprehensible.

The occurrence of jaundice may also lead to diagnostic difficulty. It may be due to hepatic injury but it may also, at times, appearing with great intensity and rapidity, be due to gas bacillus infection of the pleura when no subdiaphragmatic injury has occurred.

Abnormal physical signs may persist for months and there may be permanent alterations on the affected side. The chest is apt to show retraction, the respiratory movement is greatly diminished and the breath and voice sounds are distant or absent.

Diagnosis.—The development of an effusion very quickly after the receipt of an injury to the chest is almost certainly due to hemorrhage. In the non-traumatic cases the presence of hemothorax may be suspected if it is known that the patient is suffering from an aneurism or caries of the rib. The only other condition which occurs so quickly is a pneumothorax the physical signs of which are entirely different.

CHYLOTHORAX

The presence of a milky or lactescent fluid in the pleural or peritoneal cavity is not a common event. The discovery is nearly always accidental and, as a rule, follows the tapping of one of these cavities for what is believed to be an ordinary serous effusion. Three types of milky-like fluid are to be distinguished: chylous, chyliform and pseudo-chylous.

1. *Chylous Fluid.*—This condition most commonly follows a trauma in which the thoracic duct has been ruptured. One-third of the cases have been attributed to this cause. It may result also from anything which causes obstruction to the lymph flow either in the thoracic duct or large lymphatic vessels. Among these causes may be mentioned thrombosis of the left subclavian vein and malignant disease of the pleura, mediastinal lymph nodes or lymphatics. Chylous effusions have been encountered also in cases of filariasis. As a rule, the effusion is unilateral but it may be bilateral and in some instances the effusion is chylous on one side and serous on the other. Over one-half of the reported cases have occurred on the right side. In the majority of cases a chylous effusion is also present in the peritoneal cavity.

The fluid is usually white and milky in appearance. In some instances it has a pinkish or rosy color due to the presence of a small amount of blood. More rarely the fluid may have a yellowish or greenish tint, giving it the appearance of a purulent effusion. It is possible that, in some instances, a true chylous effusion has been mistaken for purulent fluid. On standing a creamy layer forms on the top but the fluid retains its milky appearance. The fluid is resistant against putrefaction. Microscopically the fat is seen to consist of minute globules which stain dily with osmic acid and Sudan III; there are very few cellular ele- nts. The specific gravity generally exceeds 1012. Examined chemi- ly the percentage of fat is usually found to be high, varying from to 4 per cent. Sugar and diastatic enzymes are frequently present.

Funk[1] states that to date 54 cases have been recorded.

2. *Chyliform Fluid.*—Although far from being common chyliform usions are more frequently encountered than the pure chylous. In great majority of instances chyliform effusions are associated with berculosis or malignant disease, especially the former. An effusion of is character, may be extremely chronic and usually develops insidiously. is apt to first manifest itself by gradually increasing dyspnea. Rarely e onset is accompanied by pain in the chest. As the effusion gradually reases in size there is often a sense of oppression in the chest which is once relieved by tapping. A marked feature of these effusions is the ndency to recur. This is quite characteristic of effusions due to malig- nt disease of the pleura and, at times, in those which are tuberculous origin.

Chyliform fluid is odorless, does not coagulate on standing and will ep for weeks without undergoing putrefactive changes. The color is ilky but at times may have a pinkish or rosy tint due to the admixture small amounts of blood. The milky appearance of chyliform fluid due to the presence of fat liberated by the breaking down of leukocytes d endothelial cells which have undergone fatty degeneration. Quincke[2] 1875, was the first to point out that there were two types: (1) those due pure chylous fluid (hydrops chylosus), and (2) those due to cells which d undergone fatty degeneration (hydrops chyliformis seu adiposus). icroscopically the fat globules are much larger and in addition there e numerous cells in different stages of disintegration.

3. *Pseudo-chylous Fluid.*—The fluid of pseudo-chylous effusions is aque and closely resembles diluted skimmed milk. The degree of acity often varies considerably at different tappings. The fluid may ve a pinkish tint. After standing for some time it appears greenish reflected light and a membrane forms on the surface. The remark- le resistance of the fluid to putrefactive changes may be due to two ctors: (1) the formation of a membrane on the surface; or (2) to the esence of lecithin. The specific gravity is low and the total solids rely exceed 2 per cent.

Pseudo-chylous effusions have been noted in association with cardiac ease, amyloid disease, cirrhosis of the liver and nephritis. In many es there is no adequate explanation. Recently attention has been ected to the occurrence of pseudo-chylous effusions associated with hritis, probably syphilitic in origin. Cases of this nature have been orted by Shaw,[3] Weber[4] and Weber and Schmidt.[5]

The milky appearance of pseudo-chylous effusions is not due to the sence of fat. Joachim[6] ascribed it to a pseudo-globulin. According Wallis and Schölberg[7] it is caused by a lecithin-globulin complex which is held in suspension by the inorganic salts present. Removal of the inorganic salts by dialysis results in the precipitation of the lecithin-globulin and the disappearance of the opalescence. The milky appearance is not due to free lecithin, fat, or a mucinoid substance.

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[1] *Medical Clinics of North America*, November, 1918.
[2] *Deut. Arch. f. Klin. Med.*, Bd. xvi, 1875, p. 121.
[3] *Proc. Roy. Soc. Med.*, Clinical Section, 1911, iv, 112.
[4] *Edinburgh Med. Soc.*, 1913, x, 348.
[5] *International Clinics*, vol. i, 1916.
[6] *Münch. med. Woch.*, 1903, l, p. 1915.
[7] *Quarterly Jour. Med.*, 1910, iii, 301.

Rarely the blood serum also presents a milky appearance. Oswald[1] states that formerly when venesection was a common practice, the occasional occurrence of milky-looking blood serum was noted but neve in association with pseudo-chylous effusions. The case reported by Weber and Schmidt is unique by reason of the fact that the blood serum which at first was normal in appearance, became milky when the ascite and pleural effusion were rapidly being absorbed. An unusual instanc of milky-like effusion has been recorded by West[2] in which the opalescence was caused by the presence of calcium phosphate.

As already stated the discovery of a milky effusion is almost invariabl accidental as the symptoms and physical signs are those of an ordinar effusion. The cause of the opalescence may be determined partly b microscopical and partly by chemical examinations.

HYDROTHORAX

Etiology.—By hydrothorax is meant an accumulation of clear sero fluid in the pleural cavity. It is a transudate and non-inflammatory i character in contradistinction to that encountered in pleural effusi which is an exudate and inflammatory in origin. Being one of the ma festations of a general dropsy, it occurs in a number of conditions, of whi chronic valvular heart disease and nephritis are the most commo Hydrothorax may occur also as the result of a hydremic condition of t blood or local stasis, or as a complication of a mediastinal growth, thoracic aneurism or cirrhosis of the liver.

Morbid Anatomy.—In renal cases the effusion is usually bilateral but if, as often happens, the heart is insufficient, the effusion may b unilateral and right-sided, or if bilateral there will be an excess on the right. In cardiac affections the effusion is so commonly right-sided or if bilateral, larger on the right side, that it has been for years a subject for speculation (Fig. 358). Hydrothorax is also apt to be one-sided in cases of mediastinal growth and aneurism.

The hydrothorax that accompanies heart disease has been recognized for many years. Why it occurs in some cases and not in others is not clear, for it is certain, as both Flint and Da Costa long since remarked, that the phenomenon bears no constant relation to the extent of the cardiac disease. "Not only are these lesions (cardiac) marked in cases in which dropsy has not occurred, but dropsy occurs in other cases i which the lesions are comparatively slight" (Flint).

The most interesting feature of cardiac hydrothorax is that the effusion in the majority of cases is limited to the right side or if bilatera that on the right is by far the largest. While it is quite possible that n single factor is responsible for this peculiarity it is certain that the pr dominance of right-sided accumulations is too constant to be accidenta This has long been appreciated and as far back as 1863 Baccelli offered t explanation that the enlarged heart, by dragging the superior vena ca nward carried with it the vena azygos major thus drawing it tightly nd the root of the lung and causing it to be compressed. More ntly Steele and Stengel championed the hypothesis that compres- of the azygos vein was effected by extension upward of the dilated t heart. Largely as a result of these contributions the azygos-vein othesis has been accepted as the true explanation.

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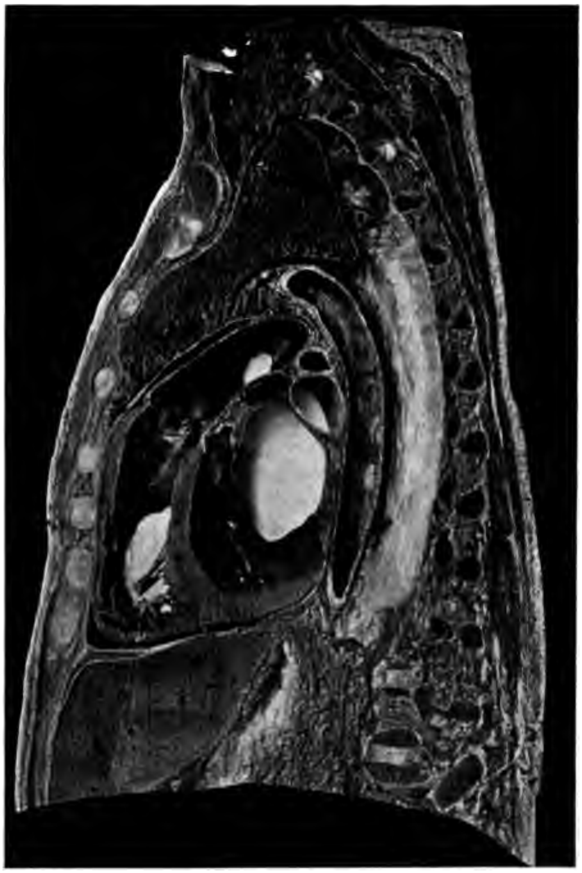
[1] "Lehrbuch der chemischen Pathologie," 1907.

[2] *Trans. Clin. Soc.*, London, 1906, xxxix, 42.



358.—Right-sided hydrothorax. Atelectasis of lung. Dilatation of left auricle.

s the result of an anatomical study of this phenomenon Fetterolf and ncluded that the azygos vein hypothesis was untenable for the fol- ng reasons: (1) Only about two-thirds of the parietal membrane is ned by the azygos vein; (2) the collateral anastomoses of the azygos s are so free and so numerous that, in the event of pressure, competent ths would soon be established and carry away any excess of fluid e azygos radicles; (3) the vena azygos minor, emptying into the major, bject to the same influences as the latter, and therefore the effusion ld always be bilateral; (4) it is anatomically impossible for the heart,

[1] Fetterolf and Landis: *Am. Jour. Med. Sc.*, November, 1909.

either directly or indirectly, to exert pressure upon the azygos major vein; (5) it does not explain purely left-sided effusions.

While dilatation of the right auricle is the deciding factor in the right-sided effusions we found that the pressure was exerted, not on the azygos vein, but on the *pulmonary veins* at the root of the lung. Confirmation of this assumption is found in the fact that Miller[1] has shown that "the capillary network into which the bronchial artery breaks up in the (visceral) pleura gives rise to radicles which join the pulmonary vein." Therefore, any obstruction of the pulmonary veins of sufficient duration and if accompanied by whatever condition of the blood essential to transudation, would cause a leakage through the visceral pleura into the pleural sac. Furthermore, the source of the pleural fluid would indicate that a similar leakage would take place into the lung itself; and of this fact there is clinical evidence that such is the case. West states that the obstruction leads to edema of the lung rather than to an effusion but that the two conditions are frequently associated. Fowler also states that in cases of general anasarca, in which unilateral pleural adhesions are present, the corresponding lung may be edematous while a considerable quantity of fluid may be found in the opposite pleura. On several occasions I have noted the coexistence of an effusion and small moist râles in the lung.

When the effusion occurs on the left side the mechanism is much the same except that the left auricle and ventricle are dilated. That the left-sided effusions are less frequent and, when present, smaller in size, is to be explained by the fact that left-sided dilatation is less frequent and also because pressure effects are less readily exerted on the left pulmonary veins.

The occurrence of a unilateral effusion as the result of a thoracic aneurism or mediastinal growth may be brought about in the same manner.

If a hydrothorax has been present but a short time the pleural surfaces show no change from the normal but if of long standing a grayish deposit may be noted over the parietal pleura. This can be scraped off readily.

The lung is collapsed but, as a rule, is readily reëxpanded on removal of the fluid (see Fig. 359). The heart as in effusions from other sources is displaced if the effusion is sufficiently large (see Figs. 109 and 206).

Symptoms.—Inasmuch as hydrothorax invariably complicates an illness which is of a serious nature, and which is characterized by well-marked symptoms, its presence is often masked. It is a notorious fact that effusions of this character are detected in the post-mortem room far more often than during life. Distressing dyspnea is so often a prominent feature in cases of failing compensation that the possibility of its being aggravated by or being entirely due to a pleural effusion is often lost sight of. It should be a fixed rule in cardiac and renal cases, to make a careful examination of the chest whenever the breathing becomes embarrassed; whenever there is a sense of oppression; or whenever the patient becomes cyanosed. Of the various cardiac affections which may be complicated by an effusion, mitral stenosis seems to be the most frequent cause. It is of the utmost importance that the fluid be removed as it often accumulates with great rapidity and of itself may be the cause of a

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[1] *Am. Jour. Anat.*, vii, 404.

termination. In any case withdrawal of the fluid affords great
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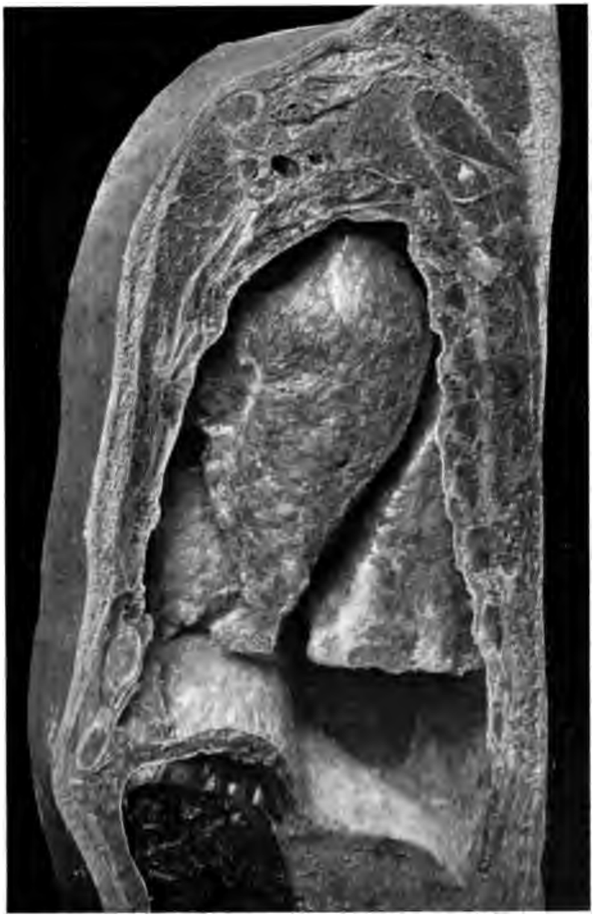
hysical Signs.—The physical findings in cases of hydrothorax are
ame as those occurring in the ordinary pleural effusion. The one
iarity of this type of effusion is that it is bilateral in about one-third
e cases. This must be borne in mind when comparing the two sides
e chest.



G. 359.—Atelectasis in a case of hydrothorax. The apex of the lung is adherent to the
tal pleura as was also the anterior surface of the upper lobe at the point at which a
fibrous scar is seen. The pleural cavity was filled with frozen serum which was re-
d. The pericardial sac is seen in front of the retracted and compressed lung. From a
itic to which infection the scar is probably due, there being no evidences of tuberculosis.

PNEUMOTHORAX

hile the term "pneumothorax" implies the presence of air or gas in
eural cavity the occurrence of either alone, for any length of time,
. In the vast majority of cases there is in addition to the air or gas

a serous or purulent effusion, hence the prefixes hydro or pyo to denote one or the other of these composite conditions. Even in cases in which nitrogen gas is introduced into the pleural cavity for therapeutic purposes the resulting pneumothorax becomes a hydropneumothorax in about 50 per cent. of the cases. Occasionally a pyopneumothorax results from this procedure; two such accidents have occurred at the Phipps Institute.

Etiology.—As an exciting cause of pneumothorax *pulmonary tuberculosis* easily stands first. One is not overestimating its importance in this connection in asserting that not less than 30 per cent. of all pneumothoraces are associated with tuberculosis of the lungs. As a complication of pulmonary tuberculosis pneumothorax can be expected in about 5 per cent. of all cases. A pneumothorax may arise also as the result of other pulmonary lesions, namely, gangrene, abscess, bronchiectasis, hemorrhagic infarct, the rupture of an emphysematous bleb, etc. Occasionally the pneumothorax is produced by an empyema rupturing into the lung and thus allowing the escape of air into the pleural cavity.

In rare instances a pneumothorax is produced as the result of the extension of a lesion in some neighboring organ as, for instance, carcinoma of the stomach or esophagus or abscess of the liver.

Perforating wounds of the chest wall may also cause a pneumo- or hemopneumothorax. Finally a hydro- or pyothorax may be changed into a hydro- or pyopneumothorax as the result of the introduction of air into the pleural cavity during the operation of paracentesis. Under these circumstances it is probably caused by faulty technique.

The condition occurs more frequently in males than females, probably because most of the statistical studies deal with hospital cases among which the males always predominate. It is almost invariably encountered in the earlier periods of life as is to be expected from its frequent association with fatal cases of tuberculosis.

Now and then one encounters an instance of so-called spontaneous pneumothorax in which the accident occurs in an apparently healthy individual and without gross disease of the lungs. Recovery is the rule.

Morbid Anatomy.—In order to appreciate the nature of this accident a knowledge of the physics of the chest is essential (see Fig. 94). "The whole outer surface of both elastic lungs is, by means of its smooth, moist covering of pleura, intimately and hermetically applied to the inner surface of the chest wall, which in its turn is covered by the parietal pleura. By reason of their complete elasticity the lungs are able to follow every change in the capacity of the thorax, without causing the two layers of the pleura ever to separate. The cavity of the unexpanded thorax is greater than the volume of the collapsed lungs when removed from the body; therefore, the lungs in their natural position within the chest must be stretched, and they are, to a certain degree, in a state of elastic tension. This tension varies directly with the size of the thoracic cavity. If the pleural cavity be opened by a perforation from without or by a wound of the lungs from within, the elasticity of the lungs causes them to collapse, and there arises an air space between the outer surface of the lungs and the inner surface of the thorax" (Landois). Furthermore, it is to be borne in mind that under normal conditions there is a negative pressure in the pleural cavity amounting to about 6 mm. of Hg. If, however, the outside air enters the pleural cavity, either as a result of rupture of the lung or perforation of the chest wall, the normal negative pressure proaches zero and as the air accumulates in the cavity the pressure bemes positive instead of negative. It is because of this elastic tension of e lungs that the heart, which is more or less movable, is held in its norıl position. If, however, one of these elastic bands is severed, as in e case of pneumothorax, the displacement of the heart toward the unected side is immediate and often attended with considerable shock. ter, as the air accumulates under pressure or an effusion develops, the splacement of the heart may be increased as the result of pressure.

Inasmuch as pneumothorax is most commonly encountered as a comication of pulmonary tuberculosis its development under these circumances will be considered in detail. In considering the pathology of ılmonary tuberculosis and of pleurisy complicating that disease it was ıown that one of the first results of the tuberculous process was an inammatory reaction in the overlying pleura which resulted in the two ıyers of the pleura becoming adherent. The older the tuberculosis the ıore dense, as a rule, is the thickening of the adjacent pleura. Without bliteration of the pleural cavity over the diseased area pneumothorax rould occur, in all probability, in a large number of cases of tuberculosis nstead of being encountered in relatively few.

In order to bring about the accident it is essential that the pleural ayers are not adherent or if they are, that the adhesions are slight and asily separated. Another requisite is the formation of a caseous area ust beneath the visceral layer, which constitutes the point of weakness. The leak may occur as the result of ulceration through the intervening barrier or a rupture of the weakened point may be produced by some unusual inspiratory strain, such as a paroxysm of coughing or severe exertion.

The occurrence of a pneumothorax is always possible in acutely advancing tuberculosis of the lungs because the disease may spread to the periphery so rapidly that the pleural surfaces have not had time to become firmly adherent. In the chronic advanced cases rupture of the lung rarely occurs over the site of the oldest disease at the apex but, as a rule, in the lower part of the lung more recently involved. For the same reason the pneumothorax may occur in the lung secondarily involved because the disease is more recent and the pleural surfaces are not so firmly adherent. In some series of cases the largest proportion of cases have occurred on the right side; in others the difference between the two sides was not marked.

In the majority of the cases the accident is encountered in the moderıtely advanced and advanced cases of tuberculosis, the rupture occurring n the lateral portion of the upper lobe between the third and sixth ribs. ccasionally, however, it is met with in incipient cases in which the ıitial lesion has been situated close to the periphery of the lung or has xtended to that location very rapidly and without producing obliteraon of the overlying pleural space.

As a rule there is but one point of rupture two or more openings may present, however. The opening may remain patulous. If it does the trapleural pressure will be found to be zero. In most instances the sues in the immediate vicinity of the perforation act like a valve which rmits the air to enter the pleural cavity during inspiration and as the spiratory movement is reversed, the valve closes so that no air can ape; as a result, air accumulates in the pleural cavity under pressure.

Unless death takes place at once or within a day or so, fluid develops.

This may be serous in the beginning, but in cases of advanced tuberculosis it soon becomes purulent.

With the development of the effusion and especially if the air accumulates under pressure, the primary displacement of the heart is increased and if the right side is involved the liver is pushed down. The displacement of the liver depends in part on the weight of the fluid and in part to the alteration of the intrapleural pressure.

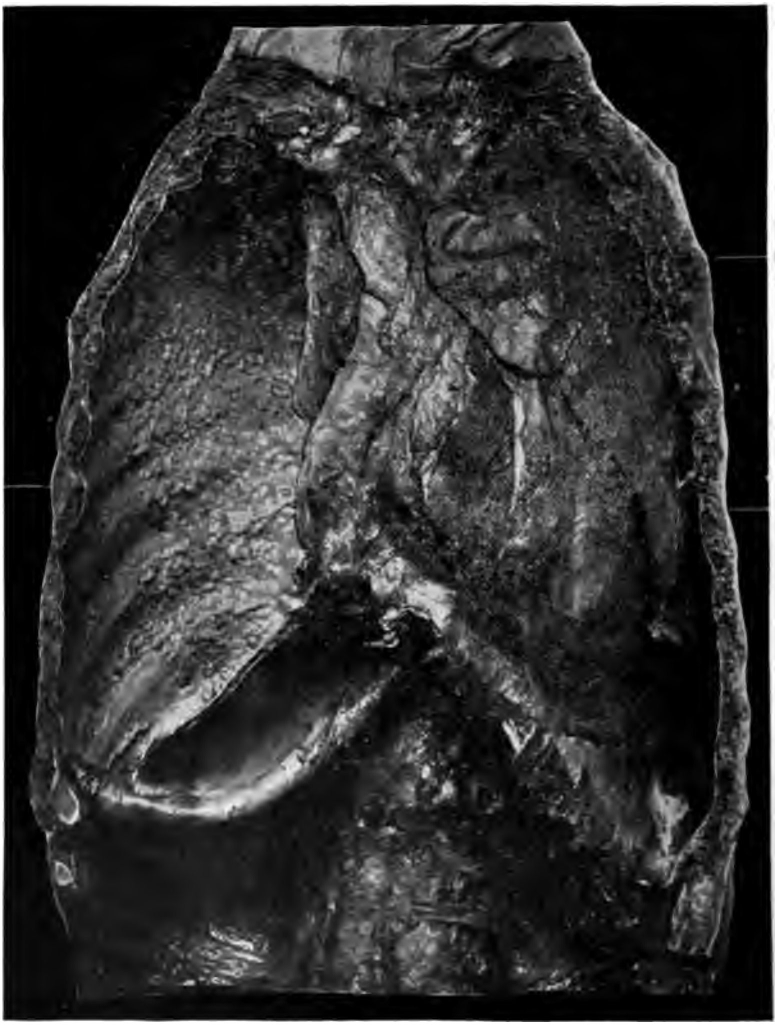
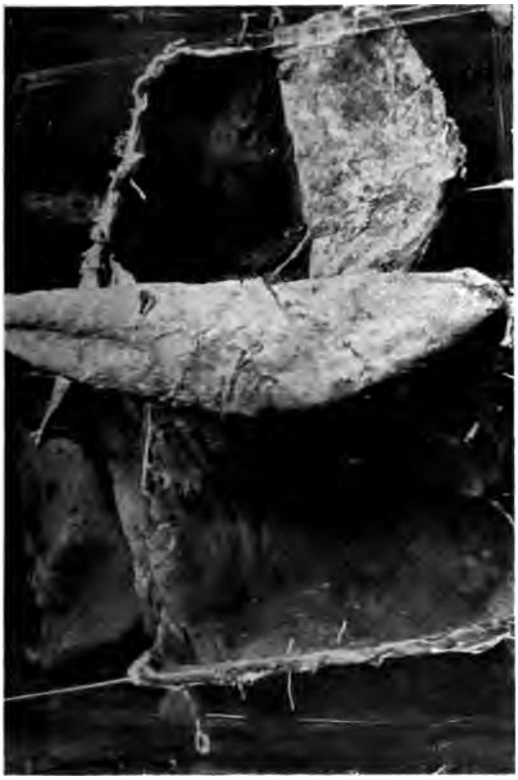


FIG. 360.—Pneumothorax with complete collapse of the right lung.

When perforation of the lung occurs the pneumothorax may be complete or partial. When complete, the entire lung is collapsed and occupies the same position as in cases of pleural effusion, namely, the upper and inner portion of the chest cavity along the spinal gutter (Fig. 360).

As the result of dense pleuritic adhesions the pneumothorax may be tial in character. Thus only the lower lobe may be collapsed (Fig.) or the air may be limited to the antero-lateral or postero-lateral ons (see Fig. 364), or it may be located in the mid-lateral region. ing the past few years roentgenologists have claimed to have seen ly large sized tuberculous cavities entirely disappear. Sampson, se and Brown[1] have shown that what had been taken for a pulmonary ity was a small *localized pneumothorax* or *hydropneumothorax* which



IG. 361.—Large cavity occupying upper half of lung. Perforation into the pleural y occurred. The thickened anterior portion of the pleura is reflected to show the ised pyopneumothorax. (*Jefferson Medical College Museum.*)

usually interlobar but might be situated between the lung and chest l. About 70 per cent. of these localized pneumothoraces occur above third rib. But often this condition is shown by the X-ray to ituated high up in the axilla, a region that is too often neglected in sical examinations. They are probably an indication of softening breaking down of the lung tissue and thus increase the gravity of prognosis. Fishberg[2] has also drawn attention to this condition also p. 627).

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[1] *Amer. Review of Tuberculosis*, 1919, vol. ii, No. 11.

[2] *Archives of Internal Medicine*, November, 1917.

The circumstances under which pneumothorax develops as a complication of other acute or chronic inflammatory affections of the lungs or of disease in adjacent organs do not require further notice. Just as in the case of tuberculosis, if the pleural space overlying the pulmonary abscess or gangrenous cavity does not become obliterated, the disease may extend through the visceral layer of the pleura and produce a pneumothorax.

Spontaneous pneumothorax may occur without gross disease of the lungs and may remain simple or develop an effusion. The fluid is serous in character and may gradually replace the air. This accident may occur as the result of rupture of an emphysematous bleb, rupture of interstitial emphysema or a tear in the lung as the result of traction of an adhesion (Fig. 362). Another variety is what has been termed *recurrent pneumothorax* in which there may be two or three attacks. The succeeding attacks may occur on the same side or first one side and then the other may be involved. The interval between attacks may be from few weeks up to several years. The majority of instances of recurrent pneumothorax are of the spontaneous type. Occasionally in tuberculosis the patient will recover from an attack of pneumothorax and later succumb to a second attack on the same or the opposite side.

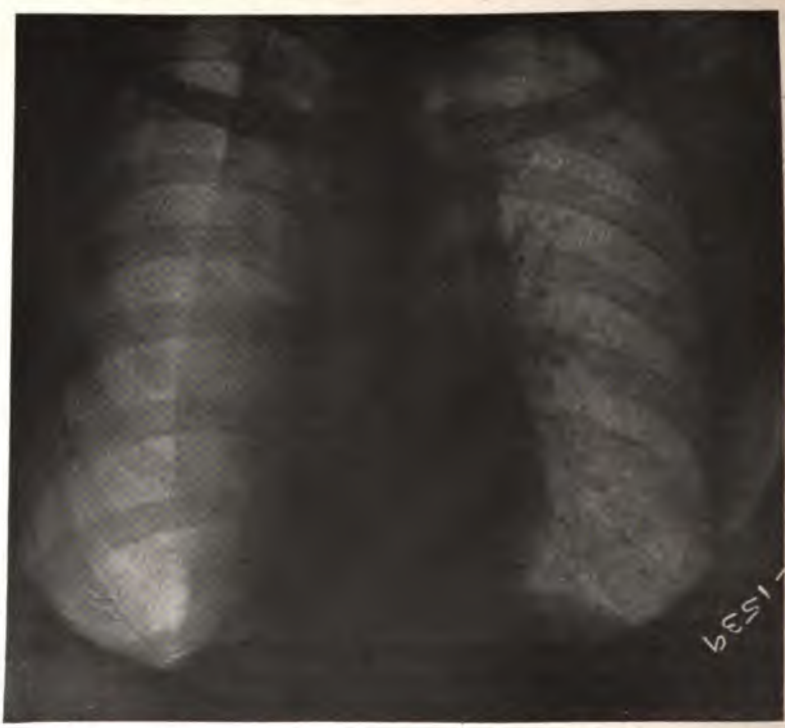

FIG. 362.—Pneumothorax. Spontaneous left-sided. (*Courtesy of Dr. D. R. Bowen.*)

Symptoms.—The onset of a pneumothorax is sudden in about three fourths of the cases. O. H. P. Pepper[1] in an analysis of 500 cases from iterature in which the onset could be determined accurately, found in 77 per cent. the onset was sudden and in 23 per cent. insidious.

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[1] *Am. Jour. Med. Sc.*, October, 1911.

s the symptomatology differs according to whether the accident is en or insidious in its onset the two types will be considered separately.

n the onset is acute, the patient is suddenly seized with an agonizing in the side which is usually felt at the costal margin. The pain is referred to as being stabbing or tearing in character. Coincidently the onset of the pain there is great difficulty in breathing. Dyspnea rhaps the most characteristic feature and is so extreme in some cases produce a feeling of impending suffocation. It is seen in its most ed form in advanced cases of tuberculosis where in addition to the psed-lung there is apt to be extensive disease on the opposite side. any cases there is every evidence of severe shock. The patient is the expression anxious, the pulse rapid and weak, the temperature the extremities are cold and the body is bathed in cold sweat. picture, however, is subject to many variations; some of the symp- may be wanting entirely or they may vary greatly as to their ity. If the patient survives, the accumulation of air under high on may increase the difficulty in breathing and in addition there is ing of oppression in the chest or distinct pain.

n cases with an insidious onset the symptoms which characterize with a sudden onset may be lacking entirely or they are so trivial o escape notice. Another thing which contributes toward mask- a pneumothorax with a gradual onset is the fact that it so frequently rs in patients with advanced tuberculous disease of the lungs. In ents of this type chest pain, dyspnea and tachycardia are nearly al- s present and may vary in severity from time to time. Under the mstances a pneumothorax might develop without attracting notice. ddition an exacerbation of one or all of these symptoms could be ibuted readily to the primary disease. The number of times that a umothorax is discovered accidently either by physical signs or an ay examination, or is first revealed at the autopsy table makes it arent that it can develop either without symptoms or with symptoms rivial in character as to escape notice.

hysical Signs.—*Inspection.*—If the patient is seen shortly after the dent he is apt to show evidence of shock and extreme dyspnea. The ure assumed is of no importance. The patient usually chooses the tion which is most comfortable; this may be lying on the back, on ound side or the affected side. If the dyspnea is marked, the patient assume a half-reclining position or sit leaning forward.

Displacement of the heart is almost constantly noted but occasionally g to the lung opposite the pneumothorax being firmly adherent to hest wall the displacement may be slight or not occur at all. Owing he rapidity of the respiratory movements and the weakness of the t's action the displacement may be difficult to determine by means of ection.

f the utmost importance is to note the character of the respiratory ements of the two sides. If the pneumothorax is complete it will en that on the affected side there is a greatly diminished respiratory rsion or an entire absence of motion. In addition the interspaces artially or completely obliterated.

f the pneumothorax has existed for any considerable length of time the unaffected side will be seen to expand more than normally as the result of compensatory emphysema on that side. In long-standing cases of pyopneumothorax the affected side is apt to become retracted as in cases of empyema, even if air still remains in the pleural cavity.

Palpation.—Absent or greatly diminished tactile fremitus is the rule. In some instances, however, the fremitus is retained. Palpation is chiefly useful in confirming the presence or absence of expansion and is also an aid in determining the position of the heart and displacement of the liver.

Percussion.—The character of the percussion note over a pneumothorax is variable. As a rule, it is tympanitic in quality and is very similar to that obtained over the abdomen when the intestines are distended with gas. By some the sound is referred to as being hyperresonant. In either case it is a full booming note.

Occasionally the air within the pleural cavity may be under such high tension as to give rise to a very high-pitched tympanitic note scarcely distinguishable from dulness. In still other instances the note may sound normal in character. These variations may be due in part to the degree of tension of the confined air and in part to variations in the sounding-board properties of different chest walls.

With the accumulation of fluid the tympanitic or hyperresonant note merges into flatness over the lower part of the chest. The existence of fluid can sometimes be determined by the presence of movable dulness.

In estimating the value of a tympanitic note on the left side it should be borne in mind that, at times, there is a very wide area of stomach tympany and which may be obtained over the antero-lateral aspect of the chest as high as the fifth rib. This error can be avoided by noting the position of the heart and also remembering that fluid is practically always present. In the latter event there would be a flat note below the tympanitic area.

Auscultation.—The breath sounds over a pneumothorax are, in the majority of cases, absent or very distant. When present, they may be of any character: broncho-vesicular, bronchial, or amphoric; and occasionally they are noted as being vesicular in character. Amphoric breathing is very suggestive of pneumothorax when heard over the middle and lower portions of the chest. It may be loud but is commonly very distant and may be missed unless carefully sought for. Occasionally the heart sounds possess a metallic quality.

As a rule, the vocal fremitus is absent or very faint. It may, however, be normal or even increased in intensity. Pectoriloquy is noted in a few cases.

Râles are not heard over the pneumothorax. Unless the lung is completely collapsed, mixed râles will be heard over the upper part of the chest, especially in the tuberculous cases.

Voçal resonance may be absent, diminished in intensity, or it may have an amphoric quality (see Fig. 57).

Metallic tinkle is a sign of great value, when present. It is a curious musical echo which accompanies the breath sounds or spoken voice or may be produced by coughing. While not always the case it usually occurs in association with amphoric breathing and amphoric voice sounds.

Succussion Splash.—This is the oldest and the most certain sign of pneumothorax. The sound is produced by the splashing of fluid in a resonating cavity containing air. To elicit the succussion splash, the patient, preferably sitting up, is sharply shaken, while the observer has his ear or stethoscope applied to the chest. If the patient is not too ill he may be made to shake himself so as to produce the sound. As in the case of other sounds produced in pneumothorax the succussion splash has a metallic, ringing quality. The patient himself can often hear the sound or feel the splashing of the liquid.

When the left side is under suspicion one must bear in mind that the gurgling sounds over the stomach have a metallic quality and are not to be confused with those occurring in pneumothorax.

The Coin Test.—Next to the succussion splash this is the most certain single symptom encountered. It is present in the great majority of cases and when heard points almost conclusively to a pneumothorax. It has been noted over a tuberculous cavity and also over a distended stomach but its occurrence under such circumstances is so rare that it may be disregarded.

If there is every reason to suspect a pneumothorax and the coin test is not at once evident the patient should be examined in both the sitting and recumbent postures, and, in addition, the coin and the stethoscope should be placed in every possible relationship to each other before a negative finding is recorded. The coin test may not appear for the first day or so following the rupture.

From February 1, 1903, to February 1, 1910, there were 473 autopsies performed in the Phipps Institute on individuals dying of pulmonary tuberculosis; among this number there were 41 instances of pneumothorax, giving a percentage of 8.6.

These cases have been analysed by Stanton[1] and Cruice.[2] The frequency of the principal physical signs noted in this series is as follows:

	Per cent.
Coin test	90.61
Expansion, absent or restricted	89.00
Hyperresonant or tympanitic percussion note	82.02
Bulging of affected side	77.04
Breath sounds:	
Distant	52.63
Absent	18.42
Amphoric	15.78
Cavernous	7.59
Broncho-vesicular	5.20
Metallic tinkle	38.88
Succussion splash	31.50

Partial Pneumothorax.—In traumatic cases and in many cases of tuberculosis the pneumothorax is complete, the entire lung being collapsed. In a very considerable proportion of cases, however, the pneumothorax is partial. This is due to the fact that the lung either is firmly adherent at some point to the chest wall or to the diaphragm. If, therefore, the lung nly anchored to the diaphragm or to the anterior or posterior aspect e chest wall it cannot completely collapse and the air may then only a part of the pleural cavity. Figs. 363 and 364 show how a dherent to the lower and lateral aspect of the chest wall cannot completely collapse. In such cases the location of the adhesion will determine whether most of the air is anterior or posterior.

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[1] Fifth Annual Report of the Phipps Institute.

[2] *Med. Rec.*, Sept. 23, 1911.

A partial pneumothorax, if sufficiently large, may manifest itself by the same symptoms as the complete variety. As a rule, however, symptoms either are entirely absent or so trivial as to pass unnoticed. If the pneumothorax is relatively large, the physical signs may be identical with those of complete pneumothorax except that the displacement of the heart is rarely as marked. In some instances the physical signs of a partial pneumothorax are masked by the pulmonary disease. This has become increasingly evident since the introduction of the X-rays in examinations of the chest. In other instances well-marked physical signs are present but are incorrectly interpreted. This was true in the case shown in Fig. 364. The patient, a young male, had been complaining of a slight cough, malaise, and slight loss of weight for several weeks. He was

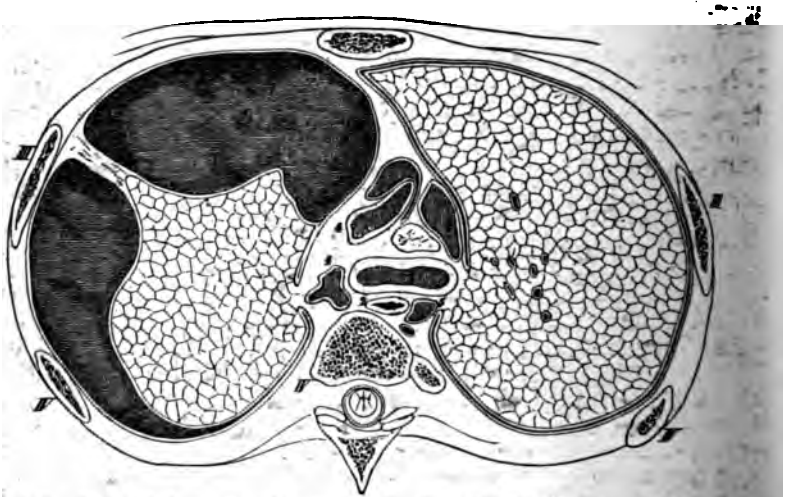
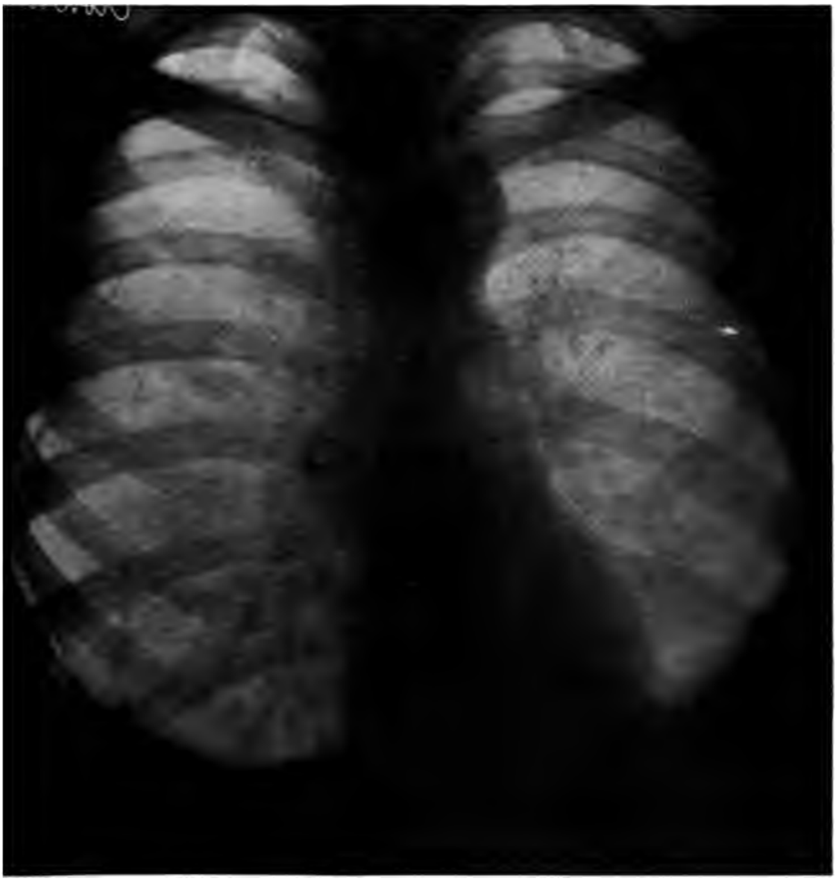


FIG. 363.—Left-sided pneumothorax. 1, Trachea. 2, Esophagus. 3, Superior vena cava. 4, Aorta. 5, Vena azygos.

Section at the level of the second intercostal space. The left lung is compressed and adherent to the costal pleura. The right lung is compensatorily large. The superior vena cava is compressed, while the mediastinal viscera are displaced toward the right. (*After Pirogoff.*)

suspected of having early tuberculosis. The examination showed restriction of motion at the right base. The percussion note over the base was slightly dull except for an area below the angle of the scapula which was tympanitic. Over the dull area the breath and voice sounds were distant; over the tympanitic area there was distinct whispering pectoriloquy and faint amphoric breath sounds. The condition seemed to indicate a chronic inflammatory condition of the left base with dilatation of the bronchi at the root of the lung. The X-ray examination showed a partial pneumothorax with the lung adherent at a point laterally and at the base. Ten days later the foregoing signs had disappeared. The breath sounds were perfectly clear except at the extreme base where the dulness on percussion and distant breathing pointed to a small effusion. A second X-ray examination confirmed these findings.

In the majority of instances a partial pneumothorax is located at some at in the lower part of the chest. I have already alluded to the fact t a small *localized interlobar pneumothorax* is not of infrequent occence in the upper part of the chest. This condition may simulate ulmonary cavity very closely. In case the patient has been under ervation the sudden appearance of cavity signs should arouse one's icion that a localized pneumothorax had occurred. Fishberg[1]



IG. 364.—Spontaneous pneumothorax on right side. Adhesion of lung to lower and al aspect of chest wall has prevented complete collapse of lung. (*Roentgenogram by H. K. Pancoast.*)

nts out that over a localized pneumothorax, situated at the apex, whispered voice is very distinctly transmitted and is often best rd or exclusively heard, high up in the axilla. In addition râles are rd over a cavity and not over a pneumothorax. In the absence of the sical signs a partial pneumothorax will usually escape detection il revealed by the X-rays.

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[1] *Loc. cit.*

Diagnosis.—Emphysema is usually cited as one of the conditions which may be confused with pneumothorax.

In cases of spontaneous pneumothorax associated with emphysema the percussion note over the emphysematous lung and the pneumothorax may closely resemble each other. Hamman[1] has called attention to the value of percussing the lower lung border. Where this is done it can be shown that resonance stops on the affected side at the level reached by the lung on the sound side in full inspiration. This point is stationary, showing no variation with inspiration and expiration. The extension of pulmonary resonance to the full capacity of the pleural space with the absence of respiratory variations at the border is absolutely characteristic of pneumothorax without effusion.

Pulmonary Cavity Due to Tuberculosis.—A large pulmonary cavity may simulate a pneumothorax very closely. The type of tuberculous case, in which one may be for a time uncertain, is that in which as the result of a chronic ulcerative process nearly the entire lung has become excavated. In extreme cases what is left of the lung is no more than a fibrous shell with a fringe of diseased tissue at the base (see Fig. 232).

Over the remnants of such a lung the percussion note is tympanitic, the breath sounds may be amphoric and the voice sounds have a metallic quality. I have seen cases of this type in which at first sight the presence of a pneumothorax seemed certain.

An error can be avoided if the following facts are kept in mind: The affected side is retracted instead of presenting a bulging appearance; the heart instead of being displaced toward the sound side is in its normal position or retracted toward the diseased side; râles can often be elicited at the extreme base where there is usually some remaining lung tissue and finally none of the classic signs of pneumothorax (coin test, metallic tinkle, succussion splash) can be elicited.

Pleural Effusion.—Occasionally a tympanitic percussion note, amphoric breathing and a metallic quality of the voice sounds can be elicited over a lung compressed as the result of an effusion. As these signs are limited to the extreme upper portion of the chest and fluid is demonstrable immediately below one should not be deceived. In pneumothorax one passes from below upward from fluid to air and thence to compressed lung tissue. Still the simulation may be very close. I recall vividly a child with empyema in which I made this very mistake.

A pneumothorax may be confused with diaphragmatic hernia, eventration of the diaphragm or subphrenic pyopneumothorax. These conditions are considered in detail in the chapter on "Diseases of the Diaphragm."

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[1] *Am. Jour. Med. Sc.*, February, 1916.

CHAPTER XXIV

DISEASES OF THE DIAPHRAGM

In spite of the fact that a knowledge of both the normal and the pathological physiology of the diaphragm is essential to a proper understanding of many thoracic and abdominal affections, references to this structure are scanty. Most of the standard text-books on medicine and many of hose dealing with diseases of the chest, either omit the subject entirely r allude to it only in a cursory fashion. Although rarely the primary eat of disease, the diaphragm is frequently involved in nearly all pulonary affections and in many of the inflammatory conditions involving he upper abdomen The neglect of the diaphragm was emphasized many ears ago by Walshe.[1] "I am persuaded," he wrote, "much of their lence depends less on the actual immunity from disease of the musculo-rous septum, than upon the rarity with which it is examined post-ortem. Clinical ignorance is the necessary result of this neglect." ecently the rôle played by the diaphragm in inflammatory affections the lungs and pleura and the upper abdomen has received more tention.

ANATOMY

The diaphragm is a great musculo-fibrous membrane, composed of uscle and fibrous tissue enclosed within two serous coats, serving as a rtition between the cavities of the thorax and abdomen. It presents double dome with the convexity toward the thoracic cavity.

In the vaults or cavities formed by the arching of the leaflets are conined on the right side, the liver, and on the left side the stomach and leen. The heart rests on the middle portion. Anteriorly the diahragm extends as high as the fourth interspace on the right side and to he fifth rib on the left side. Posteriorly the right arch extends to the evel of the angle of the scapula (eighth rib) while the left arch lies 1 nch lower (eighth interspace). The central tendon reaches the level of the eighth dorsal spine. Peripherally the diaphragm arises from the posterior surface of the ensiform; from the deep surface of the lower six rib cartilages on each side by the fleshy bands which intermingle with those of the transversalis abdominis; and from the lumbar vertebræ, the crura and the arcuate ligaments.

The main nerve supply of the diaphragm is through the phrenics. A arrow portion of the outer rim and to a slight extent the pleural and eritoneal coverings also receive branches from the intercostals (sixth to velfth).

The diaphragm is exceedingly rich in lymphatics and through these ssels the communication between the abdominal and thoracic cavities is free. So close is the connection that infective processes may pass readily through the diaphragm from the one side to the other. A knowledge of this fact is important clinically, as it will often make clear the pulmonary symptoms and signs which not infrequently follow abdominal lesions or operations, particularly those in the right upper quadrant.

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[1] "Diseases of the Lungs," 4th ed., 1871.

PHYSIOLOGY

Although the muscular action of the diaphragm closely resembles that of the heart in its rhythmic repetition, it is to be looked upon as being both a voluntary and an involuntary muscle. Under ordinary conditions it performs its work without conscious effort on the part of the individual. Its action can, however, be voluntarily arrested, increased, or checked in any part of its motion.

The normal action of the diaphragm is as follows: During the inspiratory phase the two leaflets contract. This causes the two dome-like projections to become flattened and as a result the thoracic cavity is enlarged downward and the epigastrium and hypochondria bulge outward. At the same time the distal, arched muscular parts become flatter and are drawn away from the chest wall to which they are closely applied during relaxation or the expiratory phase. The middle portion, upon which the heart rests, takes no considerable part during quiet respiration; but during forced breathing it is depressed also, to a certain extent.

The diaphragm stands in the same relation to the lower surfaces of the lungs as the chest wall does to other portions of their surface. One of the chief functions of the diaphragm is to keep the lower parts of the lungs fully expanded. If this fails and especially if the lung be forced upward by the unopposed abdominal muscles, the lower part of the lung will collapse just as failure of any portion of the chest wall to expand will lead to collapse of the underlying lung. This is seen clinically, at times, as a post-operative condition. As a result of paralysis of the diaphragm massive collapse of one of the lower lobes occurs (see Atelectasis).

The muscular action of the diaphragm differs somewhat from that of other muscles for its duration is from four to eight times that of the latter. It is, therefore, to be regarded as a tetanic movement of short duration (Landois). Of the ordinary muscles of respiration the diaphragm is the most important, particularly with reference to the part it plays in enlarging the capacity of the thorax.

In the recumbent posture (especially in thin-chested men) with the light falling obliquely on the side of the thorax, the contraction of the diaphragm can often be seen directly in the form of a wave-like movement beginning in the sixth interspace and descending one or two interspaces. This contraction wave, known as Litten's sign, is caused by the peeling off of the diaphragm from the sides of the thorax. The sign is of some diagnostic value. For a more detailed account of Litten's sign see page 32.

Examined by means of the fluoroscope, the two sides of the diaphragm, under normal conditions, are seen to rise and fall in unison. At the end of expiration the dome on the right side is usually higher (about one inch). Not only is the fluoroscope of value for determining the motility and contour of the two leaflets but in addition it enables us to inspect two important spaces. One lies between the pericardium and the diaphragm and is called the phreno-pericardial angle; it is usually mor marked on the right side. The other space lies between the outer edg of the diaphragm and the costal surface. This is known as the phreno-costal space or sulcus (Fig. 365). These two spaces disappear durin expiration but during inspiration and flattening of the diaphragm the reappear. The obliteration of these spaces, especially the phreno-costal points to adhesions or immobility of the diaphragm, or both. The actio of the diaphragm is much more pronounced in men than in women, although occasionally, as in the case of trained singers, the diaphragmati action in women is similar to that of men. Furthermore, in athleti men the excursions of the diaphragm are much greater than in those o sedentary habits.

Rarely marked muscular hypertrophy may occur as the result of overwork caused by long standing abdominal distention (Fig. 366). Rolleston[1] refers to a patient in whom marked hypertrophy of the diaphragn was found in association with cirrhosis of the liver and for which th Morison-Talma operation had been performed.

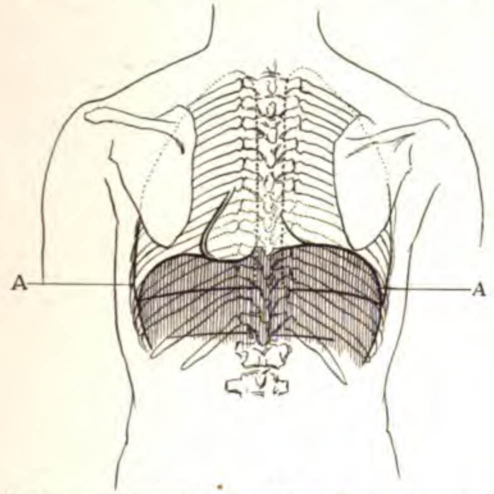

Fig. 365.—Diaphragm. Posterior view. *A A*, indicate phrenocostal spaces which ar frequently obliterated by adhesions. Heart is seen resting on the diaphragm. Th phrenopericardial angle (more marked on right side) is also frequently obliterated b adhesions.

The nerve supply of the diaphragm is through the phrenic nerve and the intercostals (sixth to twelfth). The great importance of th diaphragm in the respiratory process can be realized from the fact tha l action of the phrenic nerves in young rabbits is followed b Landois). A knowledge of the nerve distribution is important a e result of inflammatory or other conditions, is not common i hragm itself. The pain is usually referred to some distant point ainful areas are often associated with hyperasthesia or hyper f the skin and tenderness on pressure (see also "Diaphragmati " p. 44, also p. 580).

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[1] "Diseases of the Liver," 1905

From a consideration of the normal and pathological physiology of the diaphragm it can be seen that this muscle is a most important factor in the respiratory act. Anything which interferes with its work, whether this be functional or organic in character, is apt to produce a more or less marked disturbance. The disturbance in its nervous mechanism has been alluded to. Of even greater importance are those conditions which produce organic changes and which often seriously cripple the diaphragm.

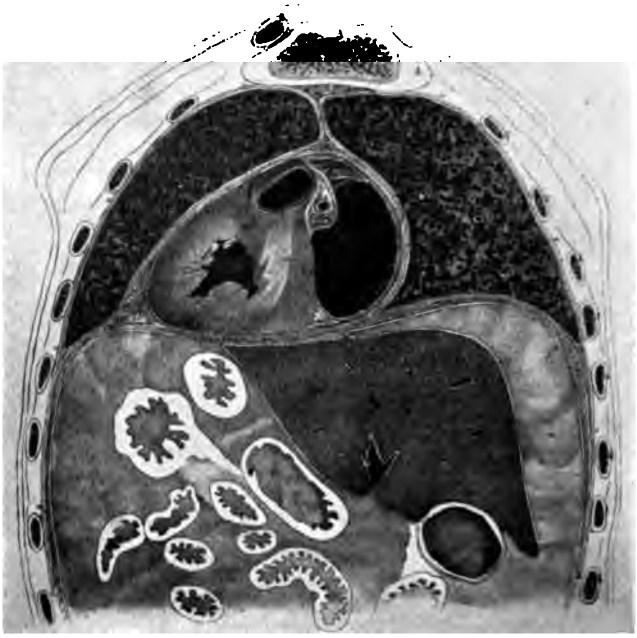

FIG. 366.—Section showing massive ascites surrounding the liver and intestines. The diaphragm is pushed upward, compressing the lungs and turning the heart into a horizontal position. (*Pirogoff.*)

FUNCTIONAL DISTURBANCES OF THE DIAPHRAGM

Inhibition.—A very common deviation from the normal is more or less inhibition of motion of one or both of the leaflets. Acute inflammation of the pleura or peritoneum is almost constantly attended by restriction in the movement of the diaphragm. In such cases it is to be regarded as protective in much the same manner as inhibition of the peristaltic movements occurs in peritonitis. In unilateral pulmonary tuberculosis the leaflet on the affected side quite constantly shows some restriction of motion even when the lesion is small and limited to the apex. This diminution in the excursion of the diaphragm on one side and its increased height are valuable diagnostic signs. The condition can be determined readily by the fluoroscope and almost equally so by noting the diminished expiratory excursion and the higher level of pulmonary resonance at the base on the affected side (see p. 350, also Fig. 259).

Spasm of the Diaphragm.—This may be either clonic or tonic in aracter.

Hiccough.—This is the most familiar result of clonic spasm. The con- ion is caused by an intermittent and sudden spasm of the diaphragm e to lesions affecting the phrenic nerves at their origin or in their urse. "The sound is produced in the larynx by the inrush of air which lows the sudden descent of the diaphragm, the glottis being, so to eak, taken unawares in a condition of incomplete dilatation" (Fowler). may recur at intervals of a minute or so or the intervals may be so ort as to temporarily interfere with breathing. If continued for an ur or so, more or less pain is apt to develop along the line of attachment the chest. In persistent cases extreme exhaustion may take place. the majority of instances hiccough is a trifling and harmless symptom ich lasts for a few minutes and then ceases. Occasionally, however, becomes persistent and uncontrollable and in such cases is to be re- rded with the greatest apprehension. It has been known to continue months, even during sleep. As one of the terminal events in fatal esses hiccough is an ominous symptom and one which often heralds proaching dissolution.

Hiccough is the expression of reflex irritation, the source of which y be above or below the diaphragm. It often occurs in acute inflam- tory conditions of the abdominal viscera, in the severe forms of hoid fever and is sometimes observed in cases of pericarditis. Its urrence in such cases is usually indicative of danger. Occasionally cough is an annoying post-operative complication particularly after erations in the upper abdomen. Hiccough and vomiting often occur the result of gunshot wounds of the diaphragm.

It is a very common symptom of indigestion. The hiccough which frequently occurs as the result of a drinking bout is of this variety. some cases the source of irritation is in the central nervous system poplexy, brain tumor, epilepsy). It may occur also as an hysterical anifestation. In many cases it is apparently idiopathic as no demon- rable cause can be found.

Paroxysmal sneezing is also said to be due to spasm of the diaphragm though the contradictory statement is made that the induction of eezing is a means of stopping the hiccough. Laughing, weeping and ughing spasms are of much the same nature as hiccough.

Tonic spasm of the diaphragm is not common. It occurs in tetanus, rychnine poisoning and hydrophobia. It gives rise to a sense of con- riction in the chest, intense dyspnea and pain along the insertion of the aphragm.

Paralysis of the Diaphragm.—This condition may be caused by a ntral or a peripheral nerve lesion. Central lesions may be due to ries or fracture of the cervical vertebræ, tumor of, or hemorrhage into e cord, progressive muscular atrophy, or myelitis. It often follows ripheral lesions in which the phrenic nerves are primarily or secondarily volved. In the former the paralysis may follow a wound in the neck ich injures the phrenic nerve. The most frequent cause of paralysis the diaphragm is diphtheria. In this disease the diaphragmatic alysis almost invariably occurs in association with other paralyses to lesions of the peripheral nerves. Any condition which leads to a re or less general neuritis, such as lead poisoning, anterior poliomyelitis, or Landry's paralysis, may involve the phrenic nerves. Paresis of the diaphragm sometimes occurs as the result of inflammation of the serous covering of the muscle.

The paralysis may be unilateral as the result of traumatic injury of the phrenic nerve on one side, or bilateral as the result of a central or peripheral lesion. The latter is by far the most frequent. When the lesion is unilateral the symptoms are not severe. There is usually some dyspnea. The severity of the respiratory symptoms will vary considerably. The base of the lung on the affected side may become congested or there may be a localized bronchitis or broncho-pneumonia. The condition should be suspected in case of a wound which would be apt to injure the phrenic nerve. Fluoroscopic examination will show the arch on the affected side to be very high.

In considering the physiology of the diaphragm it was pointed out that this muscle is the direct expander of the lower lobes. Paralysis of the muscle may cause, therefore, collapse of this portion of the lungs. Pasteur[1] has called attention to the occurrence of massive collapse of one of the lower lobes as a post-operative sequel. The collapse may be brought about in some cases as the result of paralysis of one of the leaflets of the diaphragm; in others Pasteur considers it to be due to reflex inhibition, caused by acute inflammation or acute pain in the immediate neighborhood of the diaphragm. The symptoms and signs of massive collapse are considered under "Atelectasis."

Bilateral paralysis is an extremely serious condition, and may lead rapidly to a fatal termination. The signs and symptoms, as given by Fowler, are as follows:

1. Reversal of the respiratory movements of the epigastrium and hypochondria. These regions recede during inspiration instead of bulging.
2. Absence of downward movement of the abdominal viscera during inspiration.
3. Increased movement of the lower ribs.
4. Dyspnea on exertion or excitement.
5. Alteration in the character of the voice and cough.
6. Loss of the compressive action of the abdominal muscles upon the contained viscera which ordinarily attends such acts as defecation, and the commencement (but not the continuance) of urination.
7. Feebleness of expiration and of such reflex expiratory actions as cough, sneezing, expectorating, are direct results of paralysis of the diaphragm; as the presence of that condition presents the previous inspiration from being sufficiently full to be followed by a forcible expiratory act.
8. Diminution of the total capacity of the thorax, owing to the increased arching of the diaphragm (see also p. 629).

The increased horizontal expansion of the lower thorax and reversal of the movement of the epigastrium and hypochondria are typical signs of paralysis of the diaphragm. Fluoroscopic examination will show the absence of contraction of the leaflets and the abnormally high position of the arches. They may extend to the third rib or even higher.

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[1] Annual Oration, Med. Soc., London, May, 1911.

DIAPHRAGMATIC HERNIA

The term diaphragmatic hernia should be confined to those instances in which the abdominal viscera are enclosed in a sac composed of all or but one of the component layers of the diaphragm. Stab wounds or other traumatic injuries which cause a rent in the diaphragm may also lead to protrusion of parts of the abdominal viscera into the thoracic cavity. As a rule, there is no sac but in some cases the muscle is torn while the pleural or peritoneal or both membranes remain intact. This condition should be termed evisceration and not hernia. Likewise the condition known as eventration, in which the abdominal viscera extend high into the thoracic cavity as the result of relaxation of the diaphragm, should not be confused with true hernia. Both evisceration and eventration will be considered separately.

Etiology.—Diaphragmatic hernia may be congenital or acquired. In the congenital cases the defect is the result of faulty development. In the early embryo the pleural and peritoneal cavities are continuous. Later they become separated by a thin membrane which extends forward between the lungs and the Wolffian bodies. This membrane is the beginning of the diaphragm, the development of which may be arrested in whole or in part. If the defect is extreme, the condition will be incompatible with life and is met with in still-born children. It is apt to be associated with other congenital defects. If the defect is small, the individual's life may be prolonged indefinitely. Rarely an individual with an extreme defect may reach adult life. Congenital deficiency of part of the lateral half of the diaphragm is usually placed posteriorly and on the left side. An extraordinary instance of this was recorded by Henry T. Bowditch.[1] A male adult who died as the result of a fractured spine was found at autopsy to have most of the left side of the diaphragm wanting; so that the stomach and greater part of the intestines lay in the left pleural cavity. The lung was compressed and the heart displaced to the right side. The condition was evidently congenital. In addition to absence of a part of one leaflet, a localized bulging or pouching due to weakness near the esophagus or xiphoid cartilage may occur. This may be the result of a congenital defect or may be acquired. In the latter instance severe straining may weaken the opening. Rolleston[2] is of the belief that in not a few cases, what is thought to be a congenital hernia is in reality an acquired defect due to a forgotten injury received years before. The condition occurs more commonly on the left side. Of 74 cases of true diaphragmatic hernia, Eppeniger[3] found 21 on the right side and 53 on the left.

The congenital variety is not apt to give rise to symptoms. The physical signs will be considered under "False Hernia" or "Evisceration."

EVISCERATION

This condition is often referred to as traumatic hernia or hernia spuria.

Etiology.—Evisceration is encountered far more often in men than in women for the reason that men are much more exposed to traumatic injuries. The left side is involved in over 90 per cent. of the cases. There are two reasons for this: First, the manner in which the injury commonly occurs. The wound is usually inflicted by a right-handed person striking at the most vital and most accessible part of his opponent, namely the left side. Second, the presence of the liver on the right side acts as a plug if an opening is made. Occasionally a part of the liver may pass through and become tightly constricted at the margin of the rent like a strangulated hernia. Of 561 cases of hernia spuria or evisceration, collected by Eppeniger 527 (95.6 per cent.) occurred on the left side and 34 (4.4 per cent.) on the right side. Green[1] has collected from the literature 123 cases in which evisceration has followed a stab wound of the diaphragm. To this number he adds six personal observations. Protrusion of the abdominal viscera into the thoracic cavity occurs in a little over 50 per cent. of stab wounds.

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[1] *Buffalo Med. Jour.*, June and July, 1853.

[2] "Diseases of the Liver," 1905.

[3] "Allg. u. Spez. Path. d. Zwerchfells," Suppl. Nothnagel's Spez. Path. u. Therp., 1911.

In addition to stab wounds evisceration may follow injury of the diaphragm as the result of gunshot wounds (a not infrequent occurrence in the recent war), severe straining, crushing accidents, or a blow on the chest. A strain, a blow or a crushing accident may simply tear the muscle, leaving the serous coverings intact. This group is the most difficult of recognition as there is no visible wound and the symptoms are often misleading.

Symptoms.—The initial symptoms are those of shock from which the patient may die immediately. Pain is rarely severe but there is usually a feeling of constriction in the chest. Dyspnea is usually severe and may follow the injury almost immediately. Not infrequently, however, it is a late manifestation. In left-sided cases vomiting is one of the most constant symptoms. In the case of gunshot wounds Keogh Murphy[2] states that the two most important symptoms are vomiting and hiccough and that other signs of perforation of the diaphragm are usually absent. Makins[3] states that perforation of the diaphragm with a small-bore bullet is of no great significance unless the course taken by the bullet is such that a more or less extensive slit results. In such cases the respirations are shallow, often accompanied by a groan or the slightest degree of hiccough on inspiration and a considerable increase in respiratory frequency. Pleurisy or peritonitis or both are apt to develop after a wound. If the patient survives he gradually recovers strength and may suffer but little inconvenience. As a rule, however, an irritating cough develops, dyspnea becomes marked on exertion, and there is pain in the chest. In addition there is more or less marked disturbance of the digestive process consisting of collicky pains, constipation and vomiting. Emaciation often occurs.

Physical Signs.—With few exceptions the physical signs are limited to the left side.

Inspection.—If a wound has been received its location is of great importance. The presence of a wound associated with diaphragmatic symptoms (vomiting, hiccough, dyspnea, etc.) may be sufficient to establish the diagnosis. In the case of stab wounds, the protrusion of a piece of omentum through the external wound is very common. Flattening of the abdomen and distention of the lower part of the chest, rtional to the quantity of viscera carried into the thoracic viscera, equently present. If the rent is large, the heart is displaced to the or upward and, owing to compression of the lung, expansion on the de is restricted.

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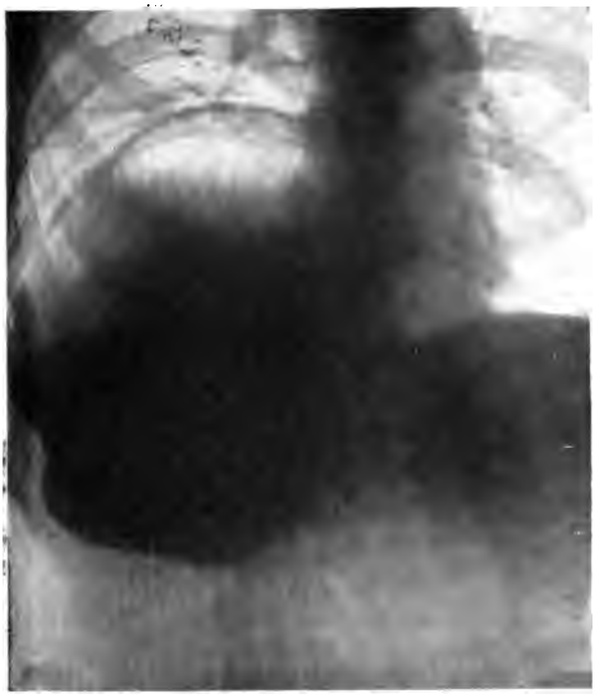
[1] *Jour. Am. Med. Assoc.*, July 15, 1916.
[2] "Wounds of the Thorax in War," 1915.
[3] "Surgical Experiences in South Africa, 1899–1900."

lpation.—This may show an absence of tactile fremitus at the left nd the displacement of the heart to the right or upward.

rcussion.—As a rule the percussion note is tympanitic at the left It may, however, be toneless owing to the displacement of the through the opening or to the presence of a hemothorax. Rarely ntestines may extend over to the right side in which case there is onary resonance above the tympany and liver dulness below.



). 367.—Traumatic hernia of diaphragm. Dark shadow below is caused by bis- Upper line of stomach can be seen through the lung shadow. (*Courtesy of Dr. H. ncoast.*)

uscultation.—Breath sounds will be absent at the base of the left and audible along the spine and upper portion of the chest. Gurg- ounds due to the movement of flatus or fluid in the displaced hollow ra may be heard.

-ray Examination.—Examined by means of the fluoroscope or graph the contour of the arch on the affected side is seen to be rmal. Becker gives the following description: The line is irregular, fairly clear at some points and blurred in others. There is usually ersal of the normal respiratory movement. If bismuth is given, pper line of the dome becomes very irregular from the new shadows;

if the stomach is distended with CO_2, the upper line extends very high and lung shadows may be seen through the distended stomach (Figs. 367 and 368).

Diagnosis.—The condition is most apt to be confused with pneumothorax, especially in those cases in which there has been no obvious wound. From the latter evisceration may be distinguished by the abdominal flattening, restriction of motion of left chest, absence of bulging of the intercostal spaces, absence of breath sounds and the presence of gurgling sounds peculiar to the stomach and intestines. Examination with the X-rays will readily distinguish the two conditions. Evisceration is also to be distinguished from eventration which is next to be considered.

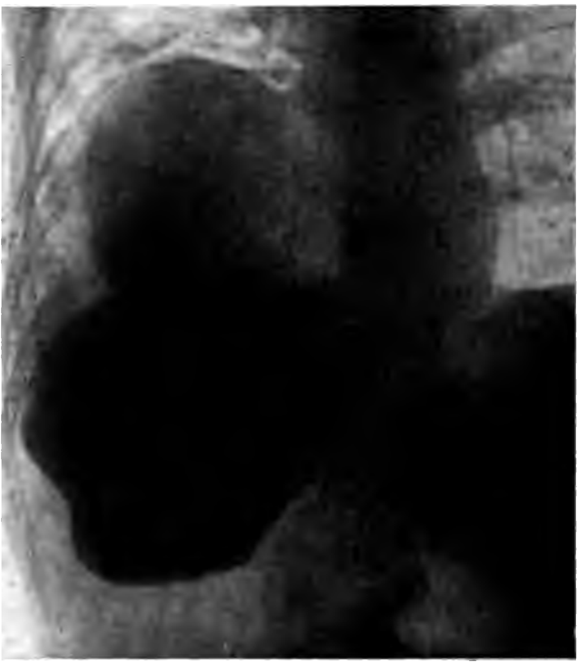


Fig. 368.—Same as Fig. 367. Showing bismuth in the stomach.

EVENTRATION OF THE DIAPHRAGM

Eventration is a relatively rare condition. It is characterized by a general expansion of one-half of the diaphragm allowing the abdominal viscera to be displaced upward into the thoracic cavity. It differs from hernia and evisceration in that the contour of the affected dome is not irregular. There is neither a bulging nor a localized opening (Fig. 369). Various names have been applied to the condition, the terms employed being more or less descriptive of what is presumed to be the anatomical factor. Thus it has been referred to as insufficiency, relaxation, dilatation, high position or elevation of the diaphragm.

Etiology.—There are two hypotheses as to the cause of the abnormally high position of the diaphragm.

1. It is believed by some to be due to increased abdominal tension long continued. There is little to support this view. An overwhelming majority of the cases have occurred in males. Women who have passed through a number of pregnancies are not predisposed to the condition in spite of the abdominal pressure. Furthermore, it has been encountered in infants and young children.

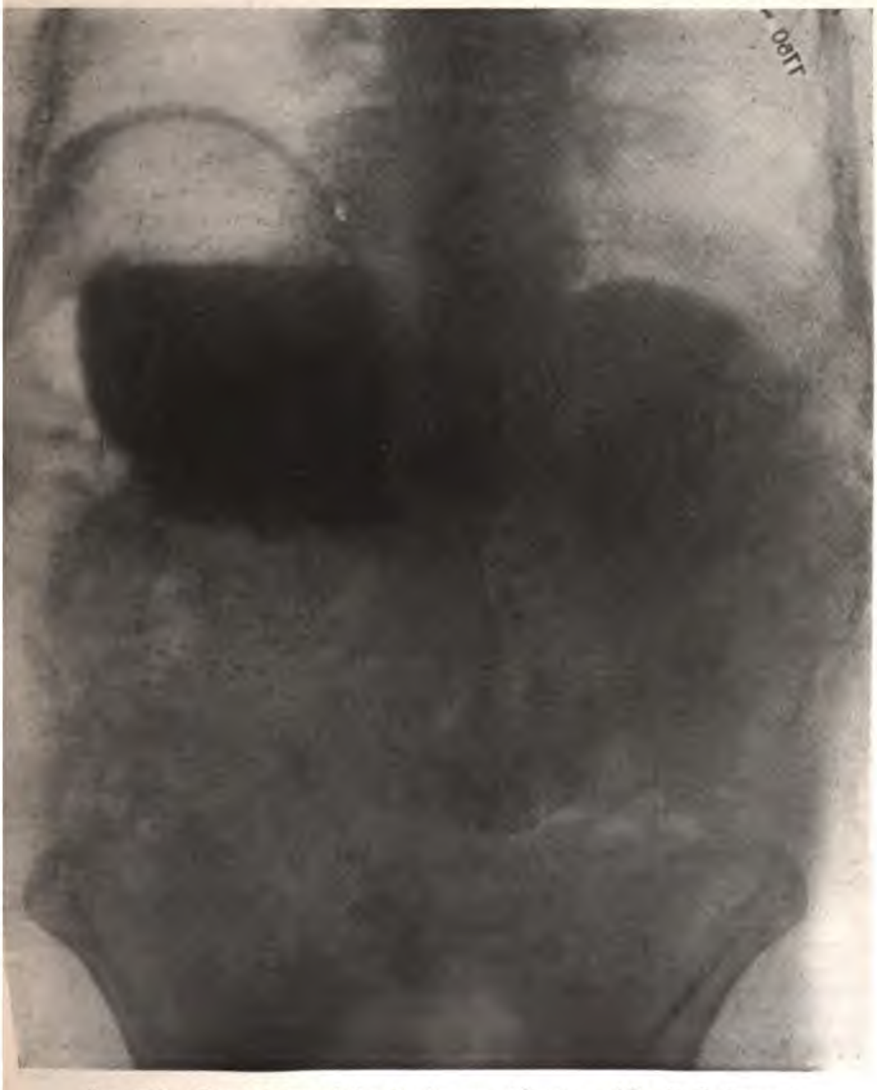


FIG. 369.—Eventration of the diaphragm. (*Courtesy of Dr. D. R. Bowen.*)

2. The weight of opinion is in favor of a congenital origin. The points in favor of this are that it does occur in infants; the lung on the affected side is not compressed; and finally there is no history of traumatism or acute infection which might lead to weakness or relaxation of the diaphragm.

It is a rare condition. In a study of 652 cases of hernia, both true and false, and eventration, Eppeniger found but 17 examples of the latter. Recently Bayne-Jones[1] reported 45 cases from the literature. His paper contains all that is known on the subject. Males are affected in the great majority of cases. Of the 45 cases so far recorded all but 3 have occurred on the left side.

Symptoms.—As the condition is congenital and the development of the thoracic and abdominal organs compensates for the abnormality, there are no symptoms. It is usually an accidental discovery which may be made in a routine physical or X-ray examination or at the autopsy table.

Physical Signs.—Of the three cases occurring on the right side the one reported by Bayne-Jones is the only one recognized during life. In this case there was absence of Litten's shadow and dulness and absent breath sounds between the third and fifth ribs. Above the dull area there was pulmonary resonance and below it tympany. Inflation of the colon displaced the dull area upward and increased the area of tympany.

In the left-sided cases there is an abnormally large area of tympany both front and back. Over this area the breath sounds are absent and there may be gurgling sounds due to the movement of fluid in the stomach or flatus. The lung is not compressed but the heart may be displaced to the right or upward.

The radiographic picture is as follows: The contour of the dome is that of a smooth, sharply defined, bow-shaped shadow with a bright area below and lung shadows above. If bismuth be given the stomach and intestines form new shadows. In a case seen by Norris (shown in Fig. 369) at the Pennsylvania Hospital the percussion note over the left chest both anteriorly and posteriorly, especially from the sixth rib downward, was very tympanitic. On auscultation the breath sounds were broncho-vesicular and in addition inconstant, muscial, splashing and gurgling sounds were heard such as usually occur in association with hyperperistalsis. The patient came to the hospital because of abdominal pain. He was later operated on for a gastric ulcer which was found to be present. Exploration on the left side revealed a very high but apparently normal diaphragm. A very long and large stomach, the left flexure of the colon and a portion of the small intestines occupied the space created by the high diaphragm.

Diagnosis.—The condition may be confused with pneumothorax, a large basal cavity, a subphrenic abscess producing gas, paralysis of the diaphragm and hernia.

The essential feature of eventration is that it is not preceded by symptoms, either acute or chronic, while all of the conditions just mentioned are, with the possible exception of a latent pneumothorax. Even in cases of latent pneumothorax the history is apt to throw some light on the condition as it is so commonly due to tuberculosis.

A large cavity would be associated with marked respiratory symptoms and other signs of thoracic damage. In subphrenic gas abscess there would be pain, fever and other evidences of sepsis. Paralysis of the diaphragm is usually bilateral. If unilateral, it is usually due to a traumatic injury of the phrenic nerve or to paresis following some acute thoracic affection, such as empyema. Hernia and evisceration both produce marked changes in the thoracic viscera (displacement of the heart and compression of the lung) and in the case of the latter there is a history of trauma.

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[1] *Arch. Int. Med.*, February, 1916.

Fluoroscopic and radiographic examinations are the most certain means of recognizing the condition.

DIAPHRAGMATIS

Inflammation of the serous covering of the diaphragm is relatively common. The pleural surface is most frequently involved and in such instances the clinical picture is fairly definite (see "Diaphragmatic Pleurisy").

Primary Diaphragmatis.—The muscle itself is rarely the primary seat of inflammatory changes. The best known example of primary inflammation of the diaphragmatic muscle is seen in *trichiniasis*. The *trichina spiralis* very frequently invades the diaphragm and in some instances sets up a serious myositis. Steiner[1] states that the diaphragm may become so extensively involved by the parasite as to produce paralysis of the muscle and death. In this connection it might be mentioned that in addition to changes in the diaphragm, respiratory symptoms are often due to invasion of the pulmonary tissue by the trichinæ. The embryos may be demonstrated in the lungs and in such cases lobar or broncho-pneumonia, hypostatic congestion or hemorrhagic areas are frequently present. Minot and Rackemann[2] in an analysis of the symptoms in 102 cases of trichiniasis found that one-half had respiratory symptoms or signs or both. In a few instances the pulmonary symptoms and signs were so prominent as to lead to a serious consideration or actual diagnosis of a purely pulmonary condition.

In the terminal stages of *scurvy* a most intense form of dyspnea and a feeling of constriction at the base of the chest sometimes occurs independently of any lesion in the lungs. In these cases there is often a brawny induration of the diaphragm or rupture of the muscle fibers and hemorrhage, identical in nature with that occurring in the voluntary muscles.

A routine examination of the diaphragm both grossly and microscopically would doubtless show that the various structures of the diaphragm are more frequently the seat of pathological changes than is at present believed. Walshe[3] states that he not infrequently noted a red, patchy, and ecchymotic discoloration of the muscle fibers; softening of the muscle or central tendon; and infiltration (serous, exudative or purulent) of the connective tissue.

Secondary Diaphragmatis.—This is a common condition and occurs far more frequently than is generally taught in the text-books.

Etiology.—The most frequent cause is an inflammatory condition involving either the thoracic or abdominal viscera. The former is by far the most important. Although croupous or broncho-pneumonia may produce inflammatory changes in the diaphragm they are, as a rule, mild and leave no permanent changes. Occasionally, however, marked hyaline degeneration may occur and in some instances so impair the function of the diaphragm as to seriously interfere with the respiratory act. The most serious secondary changes in the diaphragm follow inflammatory conditions of the pleura. The primary trouble in the pleura may be a simple, dry pleurisy, a serofibrinous pleurisy or empyema. Effusions, especially if purulent, are especially apt to damage the diaphragm. Pneumoconiosis is a common cause of immobility. The frequency with which the pleura becomes the seat of inflammatory changes in tuberculosis makes the latter disease a most important factor in changes involving the diaphragm.

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[1] *Boston Med. and Surg. Jour.*, 1908, cvi, 721.

[2] *Am. Jour. Med. Sc.*, October, 1915.

[3] Diseases of the Lungs, 4th ed., 1871.

Aside from noting the motility and position of the diaphragm, little has been done toward ascertaining the changes which occur in the muscle in tuberculous cases. Of nine cases of acute and chronic pulmonary tuberculosis in which microscopic studies of the diaphragm were made, all of them showed the presence of miliary and conglomerate tubercles; in two the diaphragm was adherent to the liver and in two there were the evidences of fibroid changes in the muscle.[1] White[2] determined the height of the diaphragm at autopsy in 187 cases of tuberculosis. In 44 instances (27 right side, 17 left side) the diaphragm extended as high as the fourth rib to third interspace. In 34 (20 right side, 14 left side) it was abnormally low, ranging from the sixth rib to the eighth interspace. Pryor[3] has reported a study of the diaphragm in 84 patients who had had an effusion. Of these cases 47 were instances of empyema; 16 of pleural effusion; and 21 of effusion associated with pulmonary tuberculosis. In the entire series the movement of the diaphragm on the affected side was normal in 14, more or less restricted in 16, and entirely lost in 54. Thus it is seen that but one out of every fourteen escaped some damage to the diaphragm. Empyema is by far the most serious condition so far as the diaphragm is concerned. As a rule but one side of the diaphragm is involved, but occasionally the condition may be bilateral.

Morbid Anatomy.—It is after all not surprising that the diaphragm should be subject to inflammatory changes when we recall its rich lymphatic supply. Through these channels bacteria may be carried upward from some point of infection in the abdomen or, what more commonly happens, the infection may occur from the lungs or pleura. It is almost inevitable that a structure bathed in the pus of an empyema will undergo inflammatory changes. The earliest change noted is a hyaline degeneration of the muscle fibers. This may be very slight and cause no permanent damage. On the other hand it may progress and eventually lead to changes in which the muscle feels like a sodden, doughy mass of infiltrated tissue. In addition it is stiff, unyielding and more or less thickened. Next, the muscle loses its elasticity and becomes quite rigid. Finally, it becomes atrophied and as the result of fibroid change, hard and immovable. As a rule the affected leaflet becomes fixed in the low or below the middle position. An additional factor in producing injury of the diaphragm in cases of effusion is the weight of fluid which has often been present for months. In the case of a large and unrecognized empyema it is no unusual thing at the autopsy table to find that the leaflet on the affected side is turned inside out, the convexity being toward the abdomen (see Figs. 345 and 346, also p. 584). This is due, in part, to loss of function and elasticity, the result of inflammatory changes, and, in part, to the weight of the fluid.

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[1] Records of the Pathological Dept., University of Penna.

[2] Fifth Annual Report, Phipps Institute, 1908.

[3] *International Clinics*, vol. ii, Twenty-sixth Series, 1916.

Associated with changes in the structure of the diaphragm are adhesions. In some instances, as in the case of dry pleurisy, the adhesions probably occur first and the chronic inflammatory changes in the muscle occur secondarily. In the case of effusions it is probable that the loss of function is primarily due to changes in the diaphragm itself and that ater adhesions form which serve to anchor the diaphragm in its abnormal position, so that even if the muscle does recover, the adhesions prevent the eaflet from asssuming its normal position and function. The portions most likely to be affected are the spaces between the pericardium and the diaphragm and the phrenocostal sulcus. The latter forming the most dependent portion of the pleural space, may retain a small amount of fluid and sediment which tend to favor adhesions even when the aspiraion has been most complete (Figs. 370 and 371). As a result of adhesions

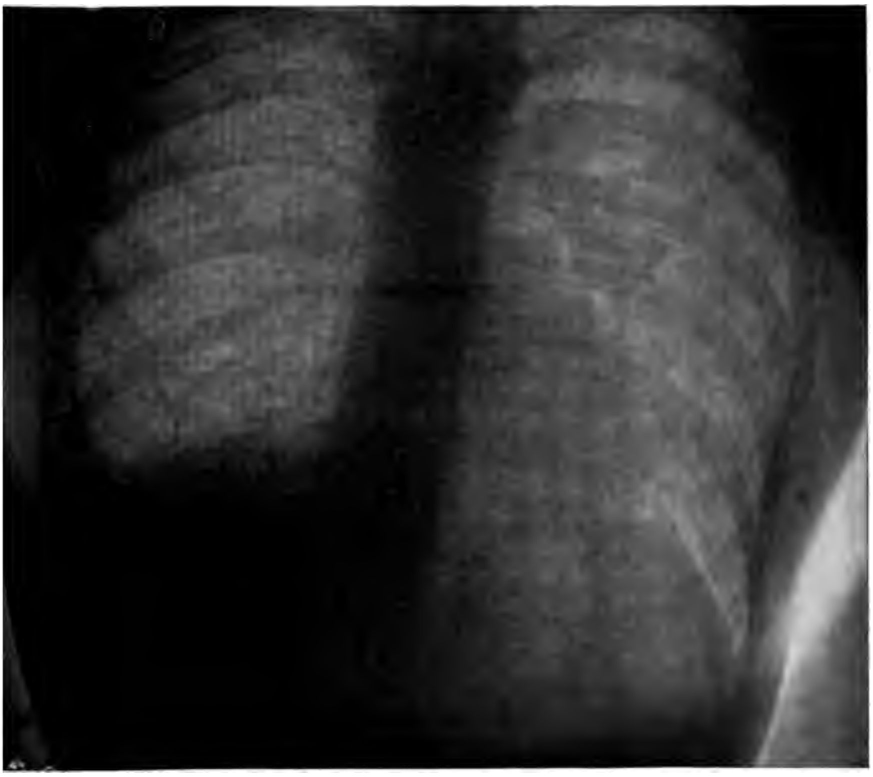

FIG. 370.—Obliteration of complementary pleural space and adhesion of diaphragm to chest wall on right side, following a pleural effusion. (*Roentgenogram by Dr. H. K. Pancoast.*)

at these points, the affected leaflet becomes more or less fixed and loses its normal contour. It may be motionless, move but slighty or one portion may show motion while another remains fixed. The heart is apt to be drawn toward the middle line and the lower lobe of the lung may become more or less atelectatic as the aspirating power of the lung upon the diaphragm diminishes or ceases.

Symptoms.—The chief symptoms of immobility of the diaphragm following inflammatory changes are dyspnea and soreness or a sense of dragging in the lower part of the chest which may persist for a long time after paracentesis. Interference with the heart's action due to adhesions may occur also. The dyspnea as a rule occurs only on exertion. In tuberculous cases dyspnea at times is very marked and is out of proportion to the amount of pulmonary damage. It is quite probable that a more careful study of these cases will show that immobility of the diaphragm is the underlying cause of the dyspnea. If both leaflets are immobile the dyspnea will be marked. Pryor[1] has reported a case in which both sides of the diaphragm were immovable and fixed in their lowest position. In a patient seen at the Phipps Institute in whom both lower lobes were the seat of a chronic blastomycotic infection there were frequent paroxysms of dyspnea which were characterized by rapid and shallow breathing and cyanosis; fluoroscopic examination showed very little diaphragmatic movement on either side and on the left side the phrenocostal sulcus was obliterated by adhesions (Fig. 372).

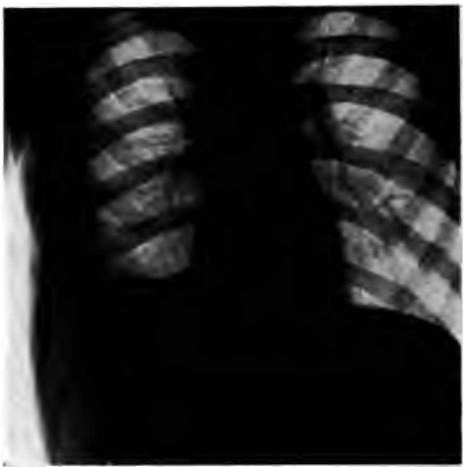

FIG. 371.—Fixation of diaphragm at outer right side following an encysted empyema Diaphragm freely movable except at point of attachment.

Physical Signs.—Immobility of the diaphragm is seen in its most characteristic form following a pleural effusion. If the effusion has been present but a few days the lung quickly expands and in the course of a few days little change is to be noted. In these cases it is probable that the diaphragm has escaped with little or no injury. On the other hand, if the effusion has been present for some time the most marked

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[1] *Loc. cit.*

ysical signs persist at the base of the affected chest in spite of the re-
val of a large amount of fluid. The usually accepted teaching has been
.t the lung has failed to expand. While this is in a measure true the
jor portion of the difficulty is to be ascribed to crippling of the functions
the diaphragm. This may be transitory in character or it may be
manent.

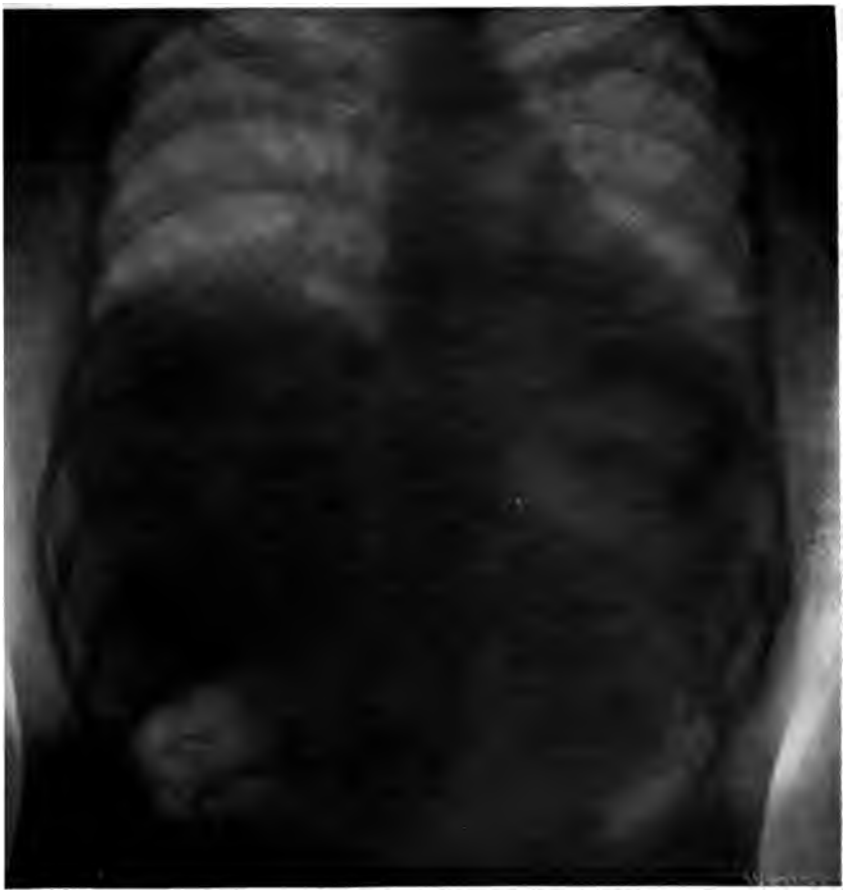

Fig. 372.—The dense shadows at both bases are due to a chronic blastomycosis. Both
ets are high and on fluoroscopic examination show scarcely any motion. Patient sub-
to frequent attacks of dyspnea. (*Roentgenogram by Dr. H. K. Pancoast.*)

Absence of Litten's sign, restriction of motion, dulness on percussion
l distant or suppressed breath sounds are the most prominent of the
ysical signs.

The most certain method of determining the condition of the dia-
agm and its functional capacity is direct inspection by means of the
oroscope or a radiograph.

In *pulmonary tuberculosis*, even when the disease is confined to a limited
a in one apex, the diaphragm on the affected side often shows a dimin-

ished inspiratory descent when examined with the fluoroscope. This is known as *Williams' early diaphragmatic sign.* Walsham and Overend[1] offer the following explanation of this phenomenon: (1) Pleuritic adhesion, at the apex, or perhaps at the base; (2) impairment of retractile pulmonary elasticity; (3) reflex inhibition of muscular action from mechanical irritation of the terminal fibers of the vagus, which might affect the corresponding half of the diaphragm, or the bronchial musculature, or possibly both; (4) the phrenic nerve fibers might be directly involved at the apex. Unilateral diminution in the inspiratory descent of the diaphragm can be determined also by percussion. This is done by marking out the base of the lung, front and back, first while at rest, and then after a deep inspiration. Even in very early cases of tuberculosis, while there is still only a small involvement at the apex, the effect upon the function of the lung of such a focus is such that there will be a distinct limitation of motion on the affected side (see p. 350).

SUBDIAPHRAGMATIC ABSCESS

(Subphrenic Abscess, Subphrenic Peritonitis)

Although entirely extrathoracic, at least in the beginning, a localized collection of pus in contact with the under surface of the diaphragm must nearly always be differentiated from analogous conditions above the diaphragm or within the thoracic cavity. Subdiaphragmatic abscess is of great interest to the student of thoracic affections not only because of the problem involved in determining the location of the pus but also because of the frequency with which the thoracic contents are secondarily implicated. Thus of 173 cases collected by Lang[2] thoracic complications were present in 140 or 82 per cent.; pleurisy, serous or serofibrinous in 41; empyema in 16; pericarditis in 16; adhesions between the lung and diaphragm and ulceration through the diaphragm in 7; perforation into the lung occurred in 34, into the pleural space in 23 and into the pericardium in 3 cases. Picqué[3] emphasizes the fact that although anatomically the abscess is below the diaphragm, it is actually within the thorax. In most cases there will be little difference in the physical signs between a localized collection of pus above the diaphragm pushing the diaphragm down and a similar condition below the diaphragm, pushing it up. It is thus apparent that subdiaphragmatic abscess is a typical example of what is known as the border-line case. The best accounts of the disease have been contributed by Rolleston[4] and Barnard.[5]

Etiology.—Clinically two types are recognized: (1) Simple diaphragmatic abscess, and (2) a gas-containing abscess, sometimes referred to as subdiaphragmatic pyopneumothorax or pyopneumothorax subphrenicus (Leyden). With few exceptions the primary focus of infection is in the abdomen and it is only occasionally that the abscess has its origin above the diaphragm. This is clearly shown in the combined statistics of Maydl,[6] Grüneisen[7] and Perutz[8] in which the primary source was intrathoracic in but 18 out of 448 cases. Generally speaking any abdominal lesion may give rise either to the simple or to the gas-containing type. The analysis of a large number of cases has shown, however, that certain conditions are mainly instrumental in forming the simple abscess and certain other conditions are most apt to lead to the gas-containing type.

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[1] *Arch. Radiology*, 1915, No. 131.

[2] Thesis, Moscow, 1895.

[3] *Revue d. Chirurgie*, 1910, xlii, 183 and 577.

[4] OSLER and MCCRAE: "Modern Medicine."

[5] Contributions to "Abdominal Surgery," 1910.

[6] "Ueber Subphrenische Abscesse," 1894.

[7] *Arch. f. klin. Chir.*, 1903, lxx, 1.

[8] *Centralb. f. d. Grenzgeb. d. Med. u. Chir.*, 1905, 8.

The most frequent cause of a simple abscess is appendicitis, either as a complication or a sequel. In a small percentage of cases appendicitis is associated with a gas-containing abscess. Elsberg[1] collected 75 cases of subphrenic abscess following appendicitis, 15 per cent. of which contained gas. Next to appendicitis the most important factor is suppuration within the liver itself. This may be a suppurating hydatid cyst, an amebic abscess or infection of the gall-bladder. Among the less frequent sources of infection may be mentioned retroperitoneal or pericolic abscess, suppuration in or about the pancreas, or the Fallopian tubes, and, rarely empyema, pneumonia or trauma. In the great majority of cases the simple abscess is located on the right side between the diaphragm and liver.

The gas-containing abscess has a different and less varied etiology. Here the most frequent cause is a perforated gastric ulcer and to a less extent a perforated duodenal ulcer. Perforation of a gastric cancer or ulcer in the colon are infrequent causes. The gas-containing abscess is more often encountered on the left side owing to the frequency of gastric ulcer as a predisposing cause. When the left side is involved the diaphragm forms the upper and posterior boundary, while the remainder of the wall is composed of some part or all of the following organs, which are commonly matted together: stomach, spleen, transverse colon and left lobe of the liver.

As already stated the conditions most likely to produce a gas-containing abscess may cause a simple abscess, and, in like manner, the conditions which predispose to a simple abscess may give rise, occasionally, to the gaseous type.

A subphrenic abscess is always unilateral, as the falciform ligament forms a natural barrier. The condition may occur at any age, even in very young children. Jopson[2] operated on a child fifteen months old in whom the abscess had developed secondarily to a pneumonia.

Lee[3] has reported six cases of *subdiaphragmatic inflammation*. The etiology was obscure and spontaneous recovery took place without suppuration. The symptoms and physical signs were those occurring in subdiaphragmatic abscess.

Morbid Anatomy.—In some instances the abscess may be considered as a localized peritonitis. As a rule, the wall of the abscess is much thickened, rough, pouched and lined with necrotic fibrin. In long-standing cases the deeper layers of the abscess wall show cicatricial tissue. In a few instances the abscess wall is smooth. The contents may consist of pus alone or pus and gas. The pus may be thick and odorless or it may be mixed with blood, bile or necrotic tissue and have a fetid odor. If the abscess is secondary to a perforated gastric ulcer it also may contain particles of food. The size of the abscess varies from one the size of a hen's egg to one which will hold one or two pints. Among the organisms found in the pus are the colon bacillus, streptococcus, staphylococcus, bacillus pyocyaneus, pneumococcus, tubercle bacillus and, rarely, the actinomyces.

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[1] *Annals of Surgery*, 1901, xxxiv, 729.
[2] *Arch. Pediatrics*, February, 1904.
[3] *Jour. Am. Med. Assoc.*, April 17, 1915.

If left to itself a subdiaphragmatic abscess often perforates the diaphragm and in this way sets up an empyema or it may rupture into the lung and become discharged through a bronchus. Rupture into the peritoneal cavity is not common. Judd[1] in reporting 36 cases from the Mayo Clinic found that in the fatal cases extension of the infection to the liver and the formation of multiple abscesses was the most frequent cause of death.

A subphrenic abscess may arise from spread of the infection by contiguity, as in the case of a liver abscess; by way of the blood stream; or through the lymphatics. Posture is an important factor, as lymphatic drainage in the abdomen is upward through the diaphragm. In addition the subdiaphragmatic space is a natural anatomical pocket when the patient is in the recumbent position, hence the importance of gravity in carrying the infection to this point. For this reason the adoption of the Fowler position by surgeons is an important factor in prevention. Barnard refers to a case in which the primary focus of infection surrounded the rectum. The infection was carried by way of the lymphatics, to the left subdiaphragmatic space where it localized and formed an abscess. It then ruptured through the diaphragm producing an empyema and eventually broke into the lung and discharged by way of the bronchi through the mouth. There are 4 cases on record in which a subdiaphragmatic abscess, caused by a gastric ulcer, ruptured through the diaphragm into the lung and was discharged through the bronchi.

Symptoms.—The onset is variable. In some cases it is sudden; in others, gradual and insidious. Those with a sudden onset are usually due to a perforated gastric ulcer. In such cases there are pain and tenderness in the left upper quadrant of the abdomen, vomiting and occasionally hiccough. In addition the patient quickly develops the symptoms of suppuration—fever, rapid pulse, quickened breathing, chills and sweating. In cases with an insidious onset the symptoms due to the abscess may develop so gradually that they may be attributed to the primary condition. If, following an operation for appendicitis, the patient has a slight elevation of the temperature, increased pulse-rate, digestive disturbances and increasing anemia attention should be directed to the right diaphragmatic region, especially if the operation wound is healthy. In some of the cases the first symptom is pain in the lower chest, usually on the right side. This may be quite severe and radiate to the shoulder or it may cause but little discomfort. Instead of the pain there may be a feeling of tenderness in the upper abdominal quadrants. All who have given much attention to subdiaphragmatic abscess emphasize the importance of suspecting the condition in individuals with indefinite signs and symptoms referable to the lower chest and in whom the symptoms of sepsis are more pronounced than can be accounted for. In the beginning respiratory symptoms are absent or slight. With secondary involvement of the thorax there is cough, and the respirations may be irregular, painful or rapid. A marked leucocytosis is the rule. If the abscess ruptures through the diaphragm and into the lung, large ities of pus may be spat up, as in the case of pulmonary abscess. e abscess is not drained, surgically or otherwise, the symptoms sis become more and more marked and the patient finally dies from stion. In such cases the duration may be from two to three months.

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[1] *Journal-Lancet*, Minneapolis, Nov. 15, 1915.

though the symptoms usually direct attention to the lower chest per abdomen they rarely indicate the location of the trouble with nce to the diaphragm. This is determined by physical examina- nd more particularly by the X-rays.

ysical Signs.—The physical signs are, in some instances, almost sively thoracic; in others, the abdominal signs will predominate; n addition they will vary accordingly as the abscess is simple or ntaining.

oracic Signs.—In simple abscess the location is, in the majority ses, on the right side and close to the spinal attachment of the ragm. For this reason the physical signs will be found over the and posterior aspect of the right lung. There is diminished ex- on on the right side, obliteration of the intercostal spaces and per- some bulging of the chest wall. Litten's sign should be looked for. ugh it is not often detected in cases of subdiaphragmatic abscess er[1] has pointed out that when the diaphragm function is impaired, because of inflammatory changes or because the leaflet is pushed s in the case of subdiaphragmatic abscess, that the costal border affected side has a greater lateral excursion than the sound side, o loss of the normal antagonism of the diaphragm to the action scaleni and intercostal muscles of the affected side. Burk[2] empha- the importance of the percussion changes. In 18 cases of right- abscess he found that the upper border of liver dulness was irregular riangular with the apex of the triangle upward in the midaxillary terior axillary line. If gas be present in the abscess, there is onary resonance, next tympany, and then liver dulness. Lee[3] ointed out that on light percussion the dulness extends as high e angle of the scapula while on deep percussion the upper limit of ss is much lower. In addition the upper limit of dulness descends ep inspiration. Tactile fremitus is usually diminished. On auscul- the breath sounds are suppressed or absent and the vocal fremitus inished. Egophony is absent. Occasionally the breathing may be hial and a friction rub may be heard. The signs are not distinctive an readily be attributed to a pleural effusion. As a matter of they may be due to an effusion, as it is not an uncommon complica- Although an exploratory puncture is of service in determining resence or absence of fluid the most valuable evidence as to the nce of a subdiaphragmatic abscess is furnished by the X-rays. ination with the X-rays shows that the dome of the diaphragm on ffected side is pushed up by a dark area and if examined with the scope the movement of the diaphragm is seen to be much restricted sent (Fig. 373). Even in cases with a complicating pleural effu- he outline of the elevated diaphragm may be made out.

a small proportion of cases a simple abscess occurs on the left side. pulmonary signs are the same. It is to be noted that the displacement of the heart is not marked and is upward rather than to the right as in cases of pleural effusion. Traube's space is obliterated.

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[1] *Archives of Internal Medicine*, Aug., 1913; Nov., 1917.

[2] *Annals of Surgery*, Oct., 1918.

[3] *Loc. cit.*

Abdominal Signs.—In some cases the signs are more noticeably abdominal although thoracic signs may be present also. In these cases the abscess is located anteriorly instead of posteriorly. On inspection there is seen to be some fulness and prominence of the right upper quadrant and the epigastrium. There is also some restriction of motion of the right lower chest. Abdominal swelling due to subdiaphragmatic abscess does not descend with inspiration because of adhesions (Barnard). The skin overlying the abscess may be edematous and red and over this area fluctuation may be obtained. Pain may be complained of in the right upper quadrant of the abdomen and on pressure tenderness is usually elicited. If the pain is referred from the chest and is due to pneumonia or pleurisy, it is usually superficial in character and not increased by deep palpation as is the case of pain which is abdominal in origin. There is usually considerable muscular rigidity in the epigastrium and right upper quadrant. The pain, the rigidity and the bulging are located in the supraumbilical region while the lower portion of the abdomen remains supple (Dieulafoy). The use of an exploring needle should be resorted to early. The X-ray findings in regard to the diaphragm are the same as in those cases in which the thoracic signs predominate.

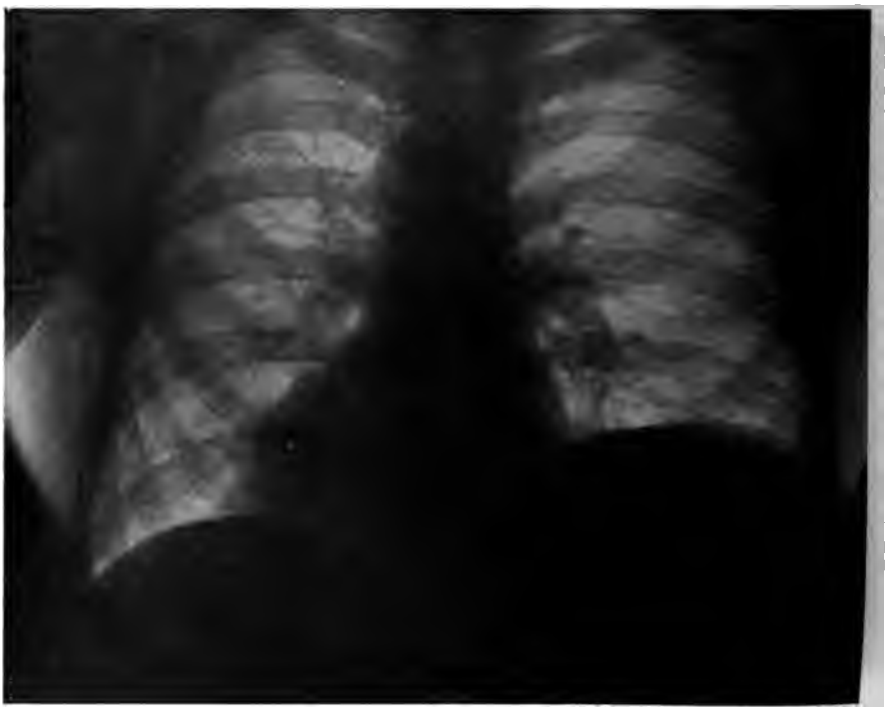

FIG. 373.—Subdiaphragmatic abscess. (*Courtesy of Dr. D. R. Bowen.*)

Signs in Gas-containing Abscesses.—(Pyopneumothorax subphrenicus).—Gas may be present from the onset or it may develop later. For this reason the physical signs may change. Owing to the frequency of perforation of a gastric ulcer as the exciting cause the left side is involved more often than the right. The physical signs may simulate those of a true pneumothorax very closely and all of the signs peculiar to the latter condition may be present, namely, a tympanitic percussion note, amphoric breathing, coin test and succussion splash. The following points may serve to distinguish the two conditions: The percussion note is tympanitic in the upper abdominal quadrant and continues upward over the chest. It never extends as high, however, as in cases of true pneumothorax. In left-sided cases the heart is not much displaced to the right but is pushed upward in the case of an abscess. When the right side is involved, the liver may be greatly displaced downward, the normal hepatic area of dulness being converted into tympany. In some cases the tympany may be confined to the area behind the sternum. Thoracic complications are almost constantly present. A pleural effusion often develops and in the beginning may be serous. The use of an exploratory needle may show the presence of a serous pleural effusion only. The abdominal signs and the symptoms of sepsis should arouse suspicion that the effusion is a secondary and not the primary trouble. A pericardial friction rub may be heard.

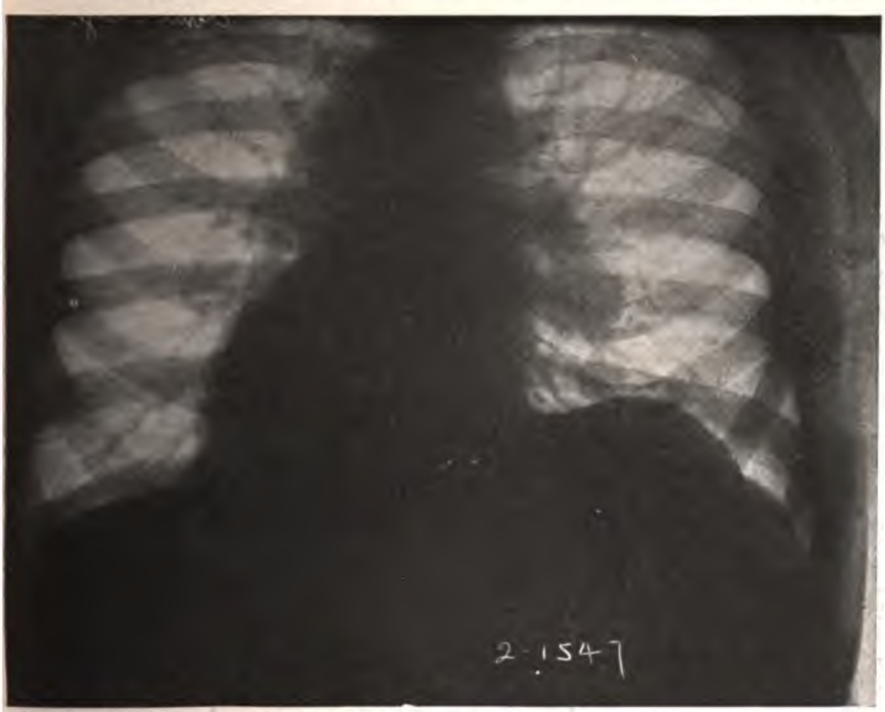

FIG. 374.—Growth or abscess of liver rather than subdiaphragmatic abscess, since there is still some diaphragmatic motion and owing to the circumscribed character of the elevation. (*Courtesy of Dr. D. R. Bowen.*)

Diagnosis.—Barnard states that the recognition of subdiaphragmatic abscess will be facilitated by attention to the following points: (I) (*a*) A history of diseases which may produce a subphrenic abscess; (*b*) the character of the onset. (II) The signs and symptoms of suppuration. (III) Abdominal signs and symptoms. (IV) Thoracic signs.

The presence of a subdiaphragmatic abscess is always a possibility when the symptoms and signs are localized in the lower chest or upper portion of the abdomen or both. An X-ray examination is invaluable and in most instances will distinguish a subdiaphragmatic abscess from conditions which give rise to somewhat the same signs. At times, however, the question may arise as to whether the bulging of the diaphragm is not due to a tumor or abscess of the liver itself (Fig. 374).

PART IV

ISEASES OF THE PERICARDIUM, HEART, AND AORTA

By H. R. M. Landis, A. B., M. D

CHAPTER XXV

DISEASES OF THE PERICARDIUM

ACUTE FIBRINOUS PERICARDITIS

Etiology.—Inflammation of the pericardium is rarely primary; it oc- rs almost without exception, as a secondary lesion or as a complication some other disease. The frequency with which acute lesions of the icardium are overlooked is illustrated by the figures given by Locke.[1] an analysis of 3683 necropsy records at the Boston City Hospital cke found 150 instances of acute pericarditis. A study of the clinical ords of these 150 cases showed that in but 27, or 17 per cent., was re any clear evidence that the condition had been recognized during .

Acute pericarditis is most commonly associated with pneumonia, sis, either local or general, and nephritis. In neither Locke's or bey's[2] experience was acute rheumatic fever an important factor. On other hand 17 of 39 cases of acute fibrinous pericarditis observed by ristian were admitted to the hospital with acute rheumatic fever or had l an attack a short time before. Robey does not attach much impor- ce to extension of the inflammation from adjacent structures. He ieves that in the great majority of cases infection of the sac occurs by y of the blood stream.

In *tuberculosis* it is not infrequently encountered either as part of general involvement of the serous surfaces, or as an independent tu- culous infection. Wolff[3] believes that a tuberculous origin should suspected in all cases of fibrinous pericarditis in the elderly not to be lained in other ways. In such cases the pericarditis is either not ompanied by demonstrable tuberculosis elsewhere or merely by healed ons in the apices. As a complication of chronic pulmonary tuberculosis icarditis is of infrequent occurrence. Among 387 autopsies at the pps Institute, tuberculosis of the pericardium was observed but 3 times.

In the chronic diseases, such as Bright's disease, diabetes and arterio- rosis a terminal pericarditis is not infrequent; it is usually latent and nmonly overlooked during life.

Pericarditis is encountered at all age periods. Males are attacked htly more frequently than females.

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[1] *Boston Med. and Surg. Jour.*, clxxv, No. 17.

[2] *Amer. Jour. Med. Sc.*, April, 1917.

[3] *Beitrage z. Klinik der Tuberculose*, 1914, xxx, No. 1.

Morbid Anatomy.—Acute fibrinous pericarditis is the mildest form of the disease. It is sometimes referred to as dry pericarditis, although there is always a slight excess of fluid present. This, however, is not demonstrable clinically. The affected area may be very small, or it may include a large part or all of the pericardial surface. The inflammation may manifest itself simply as a lusterless, roughened spot with a thin fibrinous coating which is readily removed. If of greater severity minute granulations appear on the surface of the pericardium. The capillaries are injected and proliferation of the endothelial and connective-tissue cells is set up. In addition the inflammatory infiltration penetrates the entire depth of the membrane. The change is seen most frequently at the base of the heart. In the beginning the inflammatory areas are patchy. In the severer forms these patches coalesce and the fibrinous coating increases.

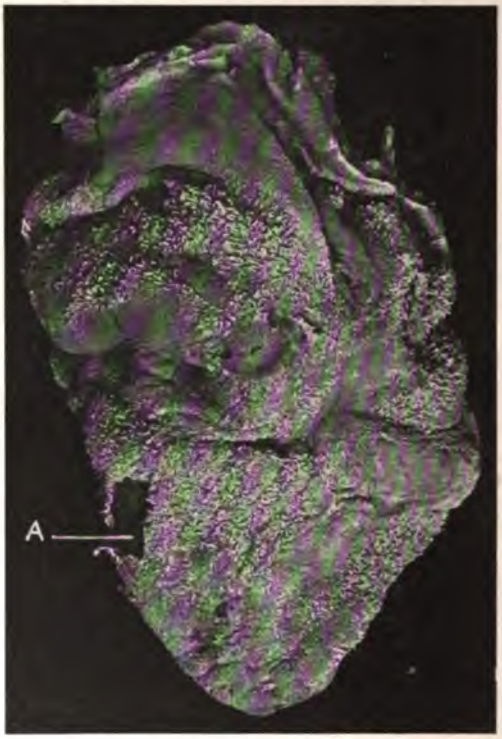

FIG. 375.—Acute serofibrinous pericarditis. The pericardium, which was opened at the bottom has been reflected upward showing both the visceral and the parietal surfaces, which are covered with thick fibrinous exudate, the thickness of which can be seen at A, at which point a small portion of the pericardium proper is exposed, the exudate having been scraped away. The illustration depicts what has been described as the "shaggy heart." (*From the Museum of the Philadelphia General Hospital.*)

In extreme cases the condition presents the so-called bread and butter appearance. Under these circumstances the heart presents a shaggy appearance, sometimes referred to as hairy heart or *cor villosum* (Fig. 375).

Symptoms.—Pericarditis, in all forms, is notoriously overlooked. In the acute fibrinous form the disease is apt to be missed, because of the nall area over which the friction rub is audible. This can be avoided ly by a painstaking auscultatory examination of the precordial region, the presence of diseases with which pericarditis is commmonly associated. nother factor which renders the recognition of the acute fibrinous form ifficult is the presence of marked pulmonary signs, which obscure the urmur produced by the friction rub. This is particularly true in cases croupous pneumonia, a disease which shows a very high post-mortem ercentage of involvement of the pericardium. This discrepancy between e bedside and autopsy findings is to be ascribed, in many instances, to e fact that acute pericarditis is frequently a terminal infection. In such ses the attention is so centered on the primary affection that painsking physical examinations are often neglected.

Pain may be entirely absent, or it may be of a very sharp, stabbing aracter. It is usually felt over the heart but may radiate into the domen near the umbilicus, to the left side of the neck or the left oulder. It is often increased by movement, deep breathing or coughg. Leaning forward in bed is a posture often assumed by patients ffering from inflammation of the pericardium, aortitis or mediastinal ease. In some cases tenderness may be elicited by pressure with the ger-tips in the region of the apex. In place of the pain there may be a ling of oppression or tension in the precordium. Slight exertion often oduces marked rapidity of the heart's action, and irregularity of the diac rhythm is common. Pain is the one symptom, however, which n be said to belong especially to pericarditis, for while the patient usually s fever and other evidences of a more or less severe illness, such manitations belong to the primary trouble rather than the inflamed perirdium.

Physical Signs.—*Inspection* is negative.

Palpation.—This is also negative except in the rare instances in which riction fremitus is felt.

Percussion.—Christian[1] has called attention to the frequency of lmonary compression signs even in acute fibrinous pericarditis with ry little or no demonstrable effusion in the sac. These signs consist an area of impairment or marked dulness over the left chest posteriorly. ne area may be small, occupying an area just about the angle of the apula or it may be one-third or one-half of the left back. In many stances there are also signs of a pleural effusion.

Auscultation.—Just as in inflammation of the pleural surfaces, ere may be no evidence of a friction rub, even when pain is marked. urthermore, while a friction rub may be produced by the slightest degree inflammatory change, it may be wanting on the other hand, in the esence of an extensive fibrinous deposit.

The pericardial friction murmur is an exocardial murmur. It is a uble or to-and-fro murmur, accompanying both the systolic and diaslic phases of the heart. The quality of the murmur suggests a rubbing grazing sound, and when loud is apt to be rough, rasping or grating character. The sound of the friction may be intensified by having e patient lean forward, or by firm pressure of the stethoscope over the lamed area. The left back, especially in the region about the angle the scapula, should be auscultated as there will often be hard onchial breathing and broncophony in association with the dulness referred to under percussion signs. Râles are not often present. Christian believes that these compression signs are due to atelectasis of the left lower lobe and that this is brought about by compression either from the heart and pericardium or from the pleural fluid or both. Inflammatory changes in the lung itself may also play a part.

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[1] *Jour. Am. Med. Assoc.*, Aug. 10, 1918.

Diagnosis.—A pericardial friction rub should not be confounded with a single endocardial murmur, or with a double aortic murmur. While it possesses some of the characteristics of a double aortic lesion, confusion can be avoided if it is kept in mind that the friction rub is limited to the precordial area; is not transmitted as are the aortic murmurs; and arterial phenomena are absent. Furthermore, while the pericardial murmur occurs during the ventricular phases it has not the same fixed relation to these phases as has the double aortic murmur.

Pleuro-pericardial Friction Murmur.—This is a comparatively common condition found in association with inflammatory conditions of the pleura, particularly those associated with pneumonia and tuberculosis. It occurs as a rule along the left border of the heart, and is caused by a roughened inflamed spot on the pleura coming into contact with a similar area on the external surface of the pericardium. In some cases the pain produced by this condition is intense. This murmur may occur during but one phase of the heart, or it may be to-and-fro. The intensity of the sound varies greatly, because it is influenced by the respiratory movements of the lungs. Holding the breath or taking a deep inspiration may cause it to disappear entirely. It is loudest when that particular period of the respiratory phase brings the two inflamed surfaces momentarily together.

TUBERCULOUS PERICARDITIS

The origin of this form of the disease is not clear. Infection by the blood stream seems the most probable because of this mode of transmission in other viscera. Extension of the disease by contiguity cannot be considered a factor of much importance in view of the relative infrequency of the disease in association with chronic pulmonary tuberculosis. Extension of the disease by way of the lymphatics is open to the same objection. Any of the anatomical forms of pericarditis may be produced by tuberculosis. In the acute form there may be a fibrinous, a serofibrinous, a hemorrhagic or a purulent exudate.

In the chronic form the pericardium may be thickened and studded with tubercles (Fig. 376). In other instances the pericardium is greatly thickened, adherent and contains varying-sized pockets of caseous material. In some instances a distinct line of caseous material may mark the line of junction. In the chronic adhesive type of pericarditis, tubercle formation is not a marked feature. Everything points to a chronic inflammatory condition in which the tubercle bacillus has played but a small part. Furthermore, the chronic adhesive type often occurs in frankly tuberculous subjects with no gross or histological evidence of tuberculosis in the pericardium. Brooks and Lippencott[1] state that among 61 instances of chronic adhesive pericarditis, tuberculosis was the apparent etiological factor in 17. In none of them, however, were tuberssions present in the pericardium, the change apparently being due oxemia of the disease rather than from direct action of the tubercle . Norris[1] found 82 cases of tuberculous pericarditis among 7219 autopsies, 1780 of which were autopsies on clinically tuberculous ials.

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[1] *Am. Jour. Med. Sc.*, December, 1909.

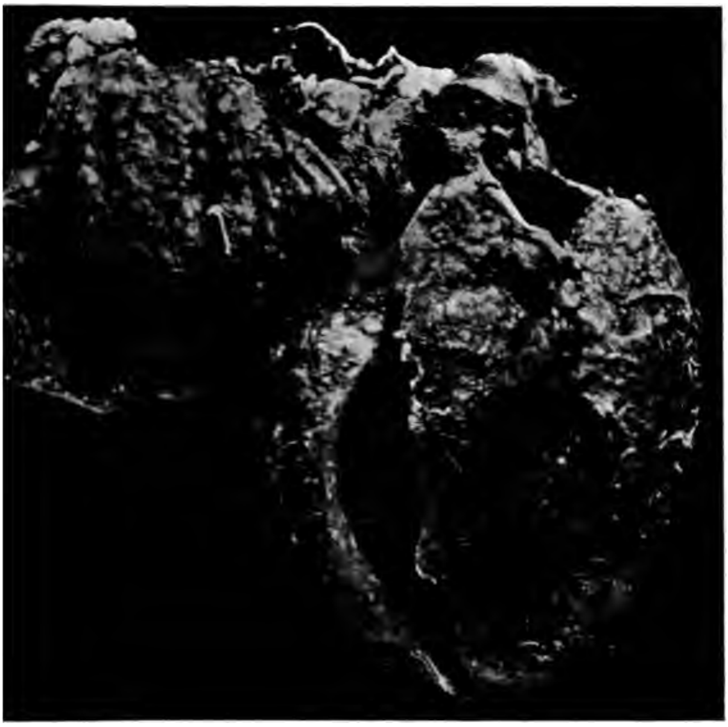

FIG. 376.—Tuberculous pericarditis.

PERICARDIAL EFFUSION

l within the pericardial sac sufficient to be designated an effusion ceed simple fibrinous pericarditis. Under these circumstances ptoms at the onset are identical with the fibrinous form. In stances there are no initial symptoms and the effusion gives no o its presence until it has attained such proportions as to produce symptoms and cardiac embarrassment. The latent forms are not ntly tuberculous or renal in origin, while those with a frank onset monly associated with rheumatic fever, pneumonia or septicemia. *opericardium.*—Actual hemorrhage into the pericardium may be stab or gunshot wound, a ruptured blood-vessel, one of the hem- diseases, malignant disease of the mediastinum or tuberculosis. ound 16 cases of hemopericardium among 8640 autopsies at the phia Hospital. Clear blood was found in the pericardium in 3 of s of tuberculosis autopsied at the Phipps Institute.

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[1] NORRIS, G. W., *Univ. of Pa. Med. Bull.*, July-Aug., 1904

Hydropericardium.—This condition is not due to inflammation but is a transudate and is, as a rule, associated with effusions into the other serous cavities. Two types are recognized, namely, the passive and the mechanical. The former is seen in association with dropsy due to Bright's disease or scarlet fever. The latter results from pressure on the large venous trunks by an aneurism of the aorta or mediastinal tumor or as the result of failing compensation (Fig. 377).

Morbid Anatomy.—To the lesions found in the acute fibrinous form is added an excessive amount of fluid, which may be serofibrinous, purulent or hemorrhagic in character. The primary disease in the serofibrinous form is usually rheumatic fever or pneumonia; in the hemorrhagic form, tuberculosis, Bright's disease, malignant disease or scurvy; if purulent in character scarlet fever, pneumonia, tuberculosis, or some septic condition such as puerperal fever, is the exciting cause (see Fig. 175).

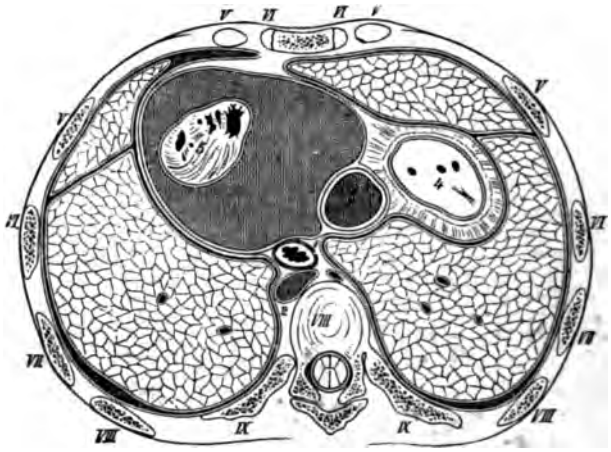

FIG. 377.—Hydropericardium. 1, Esophagus. 2, Aorta. 3, Inferior vena cava. 4, Liver. 5, Heart. (*After Pirogoff.*)

Symptoms.—If the effusion supervenes upon an attack of acute fibrinous pericarditis its recognition is sometimes facilitated by the history of precordial pain and the presence of a friction rub. If, however, pain has been wanting, and the friction rub has escaped detection, or the effusion has developed insidiously, as not infrequently happens in tuberculous cases or Bright's disease, its detection is notoriously uncertain. In the presence of one of the commoner etiological factors, such as acute rheumatism or acute croupous pneumonia, the gradual evolution from a dry fibrinous pericarditis to pericarditis with effusion may be traced readily. If, however, the case is seen after the effusion has taken place, as commonly happens, the recognition of the condition is extremely difficult. It is probably no exaggeration to state that more instances of this type escape recognition than are correctly diagnosed.

In the absence of pain the patient may complain of a sensation of discomfort in the region of the heart or of a sense of weight. Dyspnea is often the most noticeable symptom. If the dyspnea is associated with pressure symptoms, attention may be directed to the pericardium, ıerwise the heart itself will be believed to be the cause of the respiratory tress. In addition to the dyspnea pressure exerted by the distended ricardial sac may manifest itself by cyanosis of the face, distention the veins of the neck, a rapid, small and irregular pulse, dysphagia, honia and an irritating cough. It can be seen that there is nothing thognomonic in these symptoms, and that a similar picture is produced other conditions. Restlessness and insomnia are commonly present, d in addition mental symptoms have been noted.

Physical Signs.—Unless the effusion has reached a considerable ıount, probably not less than half a pint, its recognition by physical ns is hardly possible, even when there is every reason to believe that ch a condition may exist. The physical signs as given below relate to a ge collection of fluid within the sac.

Inspection.—An individual suffering from a pericardial effusion sufient to embarrass the heart's action is apt to present an anxious expresn, some duskiness of the face and difficulty in breathing. The latter is anifested by the rapid and labored character of the respirations and e posture. The patient seeks relief of his respiratory distress by having e upper part of the body raised, or by sitting up and leaning forward.

In children a noticeable bulging in the precordial region is often to be served. In adults, however, this is rarely the case, although careful pection may reveal some fulness of the intercostal interspaces (third d fourth) and possibly an undue prominence in the epigastric region. lema of the chest wall may be present if the effusion is purulent.

The apex beat of the heart is either absent or the cardiac impulse is listinct, and seen only in the third and fourth interspaces to the left.

If the effusion attains any considerable size, it so fills the thoracic vity as to cause compression of the left lung, this being apparent from diminished expansion of the left chest (Fig. 378). Normally the stal margins are drawn outward during inspiration. Hoover has pointed t that in case of a pericardial effusion the left border, from the ensiform rtilage to the eighth costal cartilage, remains stationary or is drawn ward the middle line.

Palpation.—Pressure may show the presence of a localized edema. ıe apex beat of the heart may be located in its normal position, or it may felt in the fourth interspace, especially in children. The friction b or part of it may persist at the base, and be felt on palpation. Flucation is practically never detected. Some observers have emphasized importance of the *pulsus paradoxus* in association with pericardial usion. Such a pulse is small, usually rapid and during each inspiration pulse becomes much weaker, or is entirely lost at the wrist.

Percussion.—If a pericardial effusion is to be recognized by physical signs will be largely through the percussion findings. It is to be borne in mind ıt the pericardium is a fibro-serous sac which envelops the heart; that it omewhat pyramidal in shape, resting on the diaphragm to which it is ached, and that above it merges into the external coats of the aorta and monary artery at the root of the heart. In the greater part of its ext, it is separate from the anterior wall of the thorax by the anterior rgins of the lung and pleural sacs, but is in direct relation with the left f of the lower portion of the body of the sternum.

If a large excess of fluid is poured out into the sac, either in the form of exudate or transudate, the sac distends, and extends beyond its normal limits. The commonly accepted opinion in regard to the percussion changes in pericardial effusion is that the area of dulness is pyramidal or pear shaped. This, however, is not a constant finding and its absence should not be relied on too much as evidence against an effusion. As a rule the pear-shaped area of dulness is associated with large effusions under considerable pressure and occurs late in the disease. As the result of experimental work on the cadaver Morris and Bader[1] have demonstrated that an increase of the dull area toward the base of the heart is the earliest percussion change and that this may be demonstrated with amounts of fluid as small as 250 c.c. They also call attention to the fact that Sibson, many years ago, pointed out that this dulness may extend as high as the second left costal cartilage or the first interspace or even above the clavicle. Sansom also maintains that whenever dulness extends above the third rib, there is a strong probability of a pericardial effusion. The transverse diameter is greatest at the fourth or fifth interspace. Dulness extending 1 to 2 inches to the right of the sternum is of considerable importance, and is known as Rotch's sign. In addition to the increase in the transverse diameter and the extension of dulness to the right, the area of dulness may extend to the left, beyond the apex beat; while theoretically the latter condition is possible and often mentioned as occurring, it is rarely noted (see Figs. 215–216). Obtuseness of the cardiac hepatic angle is often referred to as a reliable sign of fluid in the pericardial sac but it is more often absent than not.

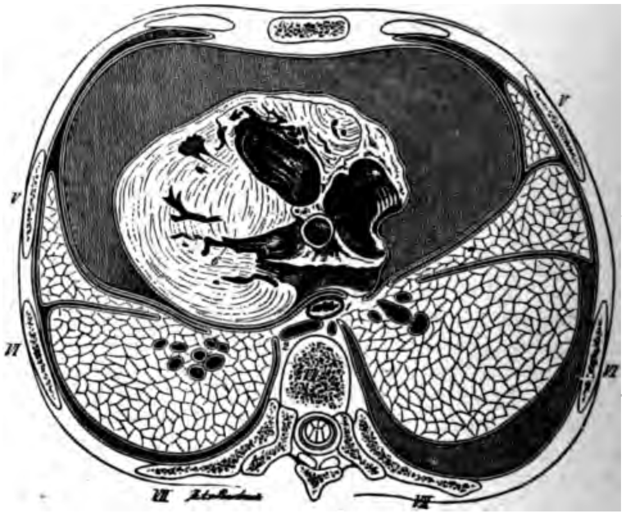

FIG. 378.—Serous pericarditis. 1, Esophagus. 2, Descending aorta. 3, Right auricle. 4, Right ventricle. 5, Left auricle. 6, Left ventricle.

The left auricle is compressed by the pericardial exudate. The antero-posterior diameter of the whole chest is increased. The heart is turned on its axis and lies more horizontally than is normally the case. The lungs are somewhat compressed and pushed far back from the anterior pericardial surface which they normally cover. The pleural sacs, however, retain their normal position, a fact which is of importance in regard to paracentesis pericardii. (*After Pirogoff.*)

If the effusion is very large, compression of the left lung may be maniested in two ways: (1) by a high-pitched tympanitic percussion note in he left axillary region; and (2) by an area of dulness posteriorly just eneath the angle of the left scapula. The latter may be increased or liminished accordingly as the patient sits up or leans forward (see lso "Compression Signs in Acute Fibrinous Pericarditis").

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[1] *Jour. Amer. Med. Assoc.*, Aug. 11, 1917.

Auscultation.—Unless the original friction rub has been heard all ver the precordium, or has been limited to the base of the heart, it isappears with the appearance of the fluid which separates the parietal nd visceral layers. In the absence of a friction rub the only noteworthy uscultatory finding is the muffled and distant character of the heart ounds at the apex, and over the body of the heart. The intensity of the eart sounds may be altered by posture, being more audible in the erect han in the recumbent position. In addition to rapidity of the cardiac ction, a disturbance of the rhythm and accentuation of the pulmonic econd sound are frequently encountered. Inasmuch as the exciting ause of endocarditis is often the same as that producing pericarditis he presence of an endocardial murmur may add to the difficulties of iagnosis.

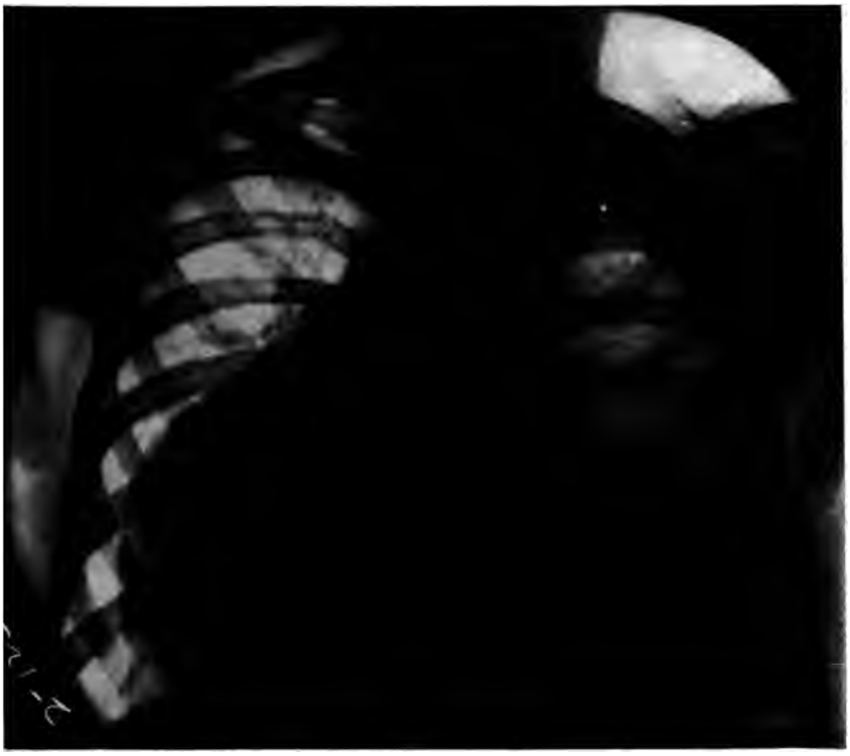

FIG. 379.—Pericardial effusion. Pleural effusion. (*Courtesy of Dr. D. R. Bowen.*)

Another associated condition which makes the recognition of pericarial effusion a difficult matter is croupous pneumonia, involving the left ng. Under these circumstances two factors aid in obscuring the secondary condition; first, the symptoms referable to the pulmonary affection; and secondly, the physical signs.

Diagnosis.—Aside from entirely overlooking the presence of fluid in the pericardial sac, the next most common error is in mistaking the condition for *cardiac dilatation.* And while the distinction between the two conditions is theoretically easy, their differentiation at the bedside is often a difficult matter. In weighing the evidence it should be remembered that in dilatation the apex beat is visible and diffuse; the area of dulness is an exaggeration of the normal triangular space and does not extend upward or assume the pear-shaped area seen in effusion, nor does it alter the percussion note over the left lung; the heart sounds are distinct, valvular or fetal in character, and gallop rhythm is common. If the patient is stout and the chest wall thick, the points of difference in favor of cardiac dilatation are greatly obscured.

Because of the strong cardiac impulse and intensity of the heart sounds simple hypertrophy of the heart should cause no confusion.

An encysted collection of fluid between the lobes of the lungs, or in the pleural sac may simulate a pericardial effusion. The differentiation will rest largely on the character of the physical findings.

A tumor in the anterior mediastinum may approximate the area of dulness occurring in a pericardial effusion. An example of this is shown in Fig. 326.

The X-ray is the most certain means of detecting a pericardial effusion and in doubtful cases should be resorted to whenever possible (Fig. 379).

PNEUMOPERICARDIUM

Laennec in his treatise on "Mediate Auscultation and Diseases of the Chest" stated that "aëriform effusions" were frequently encountered in the dissection of bodies which had been kept for some time. This he ascribed to putrefaction in some cases but in many others he thought the air or gas had its existence previously to death. This observation is discredited by all modern writers and pneumopericardium is now admitted to be an extremely rare condition. In 1904 James,[1] in reporting a personal observation, found references to but 37 authentic cases in the literature. Ten years later Cowan, Harrington and Riddell,[2] added 6 more cases. In addition to these I have found references to 2 more cases, making a total of 46 up to January, 1919. This represents, in all probability, the total number. In spite of the fact that it has never fallen to the lot of any one man, with the exception of Mueller, to see more than one example of this condition, it is worthy of mention that 40 of the 46 cases were recognized as instances of pneumopericardium before death. The fact that the nature of the trouble should have been readily recognized by observers who were meeting it for the first time speaks strongly for the uniqueness of the acoustic phenomena.

Etiology.—In 34 of the cases there was an accidental communication between the pericardial sac and some neighboring viscus; in 8 no such communication was apparent at the autopsy. It is possible the that gas may form in the pericardial sac as the result of disintegrat a foul exudate or that it may arise as the result of infection by a lucing organism, although such an instance is yet to be recorded. te pericarditis was the exciting cause in 2 cases (Stokes, Ljungdahl). cer of the esophagus was present in 5 cases; in 2 the esophagus and cardial sac were perforated by a foreign body, once by a fish-bone nes) and in the unique case recorded by Walshe, a juggler caused a oration in attempting to swallow a sword. In several cases the initial on, usually a gastric ulcer, perforated through the diaphragm into the cardial sac. Tuberculous ulceration of the lungs, gangrene of the g and empyema have also been cited as causes of a perforation. Paratesis of the pericardial sac (2 cases) or of the pleural sac (1 case) have n the exciting causes in three instances. In a traumatic case reported Burke[1] it was noted at the operation that during the diastole of the rt air bubbles appeared through the small slit in the sac which had n caused by a stiletto.

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[1] *American Medicine,* July 2, 1904.

[2] *Quarterly Jour. Med.,* 1914, vol. vii.

Morbid Anatomy.—In the majority of instances the lesion which gives to the perforation infects the sac so that an effusion also occurs and, a rule, this sooner or later becomes purulent. In the case reported by nes the two layers of the pericardium were deeply fissured and preted a leathery and corrugated appearance. They were covered with hin layer of foul-smelling greenish, sticky pus.

Symptoms.—Rarely, as when an acute pericarditis is the exciting use, the onset is gradual. In such cases the symptoms at first will be se of simple pericarditis. Later when the distention has become at, pressure symptoms on neighboring organs and functional disturbces of the heart will occur. If, however ,perforation has been produced, her by erosion or a trauma, the onset is sudden. The patient is ocked and cyanosed and complains of shortness of breath, precordial in or oppression and palpitation. The pulse is small and weak, and ere may be fainting attacks. Dysphagia due to pressure on the esophas has been noted also. The clinical picture is not unlike that seen pneumothorax (McPhedran).

Physical Signs.—These are striking and characteristic, so markedly that they almost invariably suggest the diagnosis although observed the first time.

Inspection.—There may be a noticeable bulging of the precordium; is may be very marked. The most important piece of evidence to be tained by inspection is the presence or absence of the apex beat and the rdiac impulse. In the recumbent position the apex beat and cardiac pulse are not seen but when the patient assumes the upright or lateral sture or bends forward they reappear. This is due to the fact that the r or gas, obeying the law of gravity, always occupies the uppermost rtion of the sac.

Percussion.—This is often sufficient to establish a diagnosis. In the cumbent position there is a high-pitched tympanitic note over the area normal cardiac dulness and extending as low as the fifth rib. When e patient sits up the upper portion of the precordium is tympanitic id the lower portion dull. With the patient lying on the right side an ea of dulness may be obtained along the right border of the sternum to yond the left border and extending from the second interspace downard to liver dulness. Stokes obtained a "cracked pot" sound in his se.

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[1] *Buffalo Med. Jour.*, May, 1909.

Auscultation.—The auscultatory signs are distinctive. In the great majority of the cases there has been heard over the precordium a rhythmic, churning and splashing sound distinctly metallic in quality. In some instances metallic tinkling or "mill wheel" sounds (the *bruit de la roue hydraulique* of the older French writers) are heard. The sound has been likened also to that made by an old-fashioned churn (Meigs). Stokes'[1] description has been quoted frequently: "A series of sounds was observable which I had never before met with. It is difficult or impossible to convey in words any idea of the extraordinary phenomena then presented. They were not the rasping sound of indurated lymph or the leather creak of Collin, nor those proceeding from pericarditis with valvular murmur, but a mixture of the various attrition murmurs with a large crepitating and a gurgling sound, while to all these phenomena was added a distinct metallic character." These metallic sounds may be louder on inspiration than on expiration and may vary in intensity from time to time without apparent reason. They may also be heard at some distance from the chest.

About one-third of the cases have recovered. In four of the recently reported cases radiographic or fluoroscopic examination has aided in the diagnosis.

Diagnosis.—Experience has shown, even from the limited number of cases on record, that there is a little likelihood of pneumopericardium being mistaken for something else or vice versa. Three remote possibilities present themselves: (1) Metallic succussion sounds may be produced by the heart in a hydro- or pyopneumothorax and in these conditions the cardiac area of dulness may be replaced by tympany. In pneumopericardium the sounds are louder and more localized and the apex beat disappears and reappears with change of posture. (2) An area of tympany to the left and beneath the heart, is sometimes present in cases of diaphragmatic abscess containing gas. The tympanitic area does not obscure the area of normal cardiac dulness in the recumbent posture nor is it above the dull area in the erect or sitting posture. (3) A large area of gastric tympany associated with metallic, rumbling sounds in the stomach. The sounds are not rhythmic nor does the tympany replace the area of cardiac dulness.

ADHESIVE PERICARDITIS

Etiology.—Chronic adhesive pericarditis may be the final stage of the acute fibrinous form, although it rarely falls to the lot of any one observer to follow this change. The adherent type is most frequently encountered years after the initial mischief, and almost invariably presents itself in some form of cardiac embarrassment. Acute rheumatic fever is the most frequent predisposing cause. In the absence of a history of this infection, tuberculosis seems to be the next most important factor. A tuberculous origin of the trouble is to be suspected, especially if the patient presents any evidence of tuberculosis, either latent or active. Brooks and Lippencott[2] in a study of 1000 protocols found 150 instances of pericarditis, 61 of which were of the chronic adhesive type. Of these ses tuberculosis was the apparent etiological factor in 17. Holst[1] l evidences of adhesive pericarditis in 61 cases among 1586 autopsies. rculosis was present in 10 of the 61 cases (see "Tuberculous Peri- tis," p. 656). Sicard among 2000 autopsies found adhesive peri- tis in 77 instances. The condition may be expected, therefore, in t 4 per cent. of all cases coming to autopsy.

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[1] Diseases of Heart and Aorta, 1854, p. 22.
[2] *Am. Jour. Med. Sc.*, December, 1909.

orbid Anatomy.—Several varieties of adhesive pericarditis are en- tered. (1) The adhesions may obliterate only a portion of the peri- al sac (see Fig. 218). (2) Obliteration of the sac may be complete 380). (3) The sac may be obliterated and in addition is adherent rrounding structures (mediastino-pericarditis) (Fig. 381). (4) As a of a multiple serositis. When old the fibrous tissue is dense and h and cannot be separated from the heart without tearing the latter. n adherent to the surrounding structures the heart, lungs and astinal contents cannot be separated and must be removed *en masse*. he heart muscle itself is apt to present degenerative changes and is very commonly an associated endocarditis. In common with c insufficiency and general arterio-sclerosis, adhesive pericarditis produces the most extreme degree of cardiac hypertrophy met When symptoms do arise they are usually due to myocardial ges which may have developed concomitantly with the pericardial ge or independently.

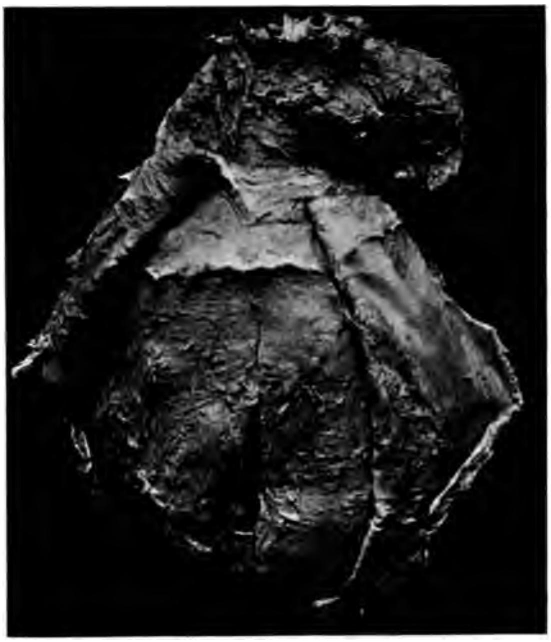

FIG. 380.—Chronic adhesive pericarditis.

athological Physiology.—The mechanical effects upon the circula- due to the adhesions are described by Hirschfelder as follows: (1)

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[1] *Norsk Magazin for Laegevidenskoben*, October, 1914, lxxv.

The work of the ventricle is increased by the tug upon the adhesions. (2) The filling of the heart may be hindered by strangulation of the vena cava. At each contraction the heart must not only drive out the blood, but must pull on its harness of adhesions. The additional work will depend on the tightness of the adhesions and the weight and rigidity of the structures pulled. Adhesions between the ribs and diaphragm cause the greatest effort. (3) The emptying of the heart and the flow through the aorta may be hindered by the tugging of adhesions upon the arch of the aorta.

As the result of the increased work the heart hypertrophies just as it does in the presence of chronic endocarditis. As degenerative changes are relatively common in the heart muscle, either as the result of the fibrosis extending in from the pericardium or as an independent affection, compensation is apt to fail if the heart is subjected to an undue strain. When compensation fails, it may present the clinical picture of failure of the left ventricle with marked dyspnea or pulmonary edema; or it may manifest itself chiefly by the signs of venous stasis such as occur in failure of the right ventricle. Hirschfelder states that the type of broken compensation will depend on whether the adhesions are greatest over the left or over the right ventricle.

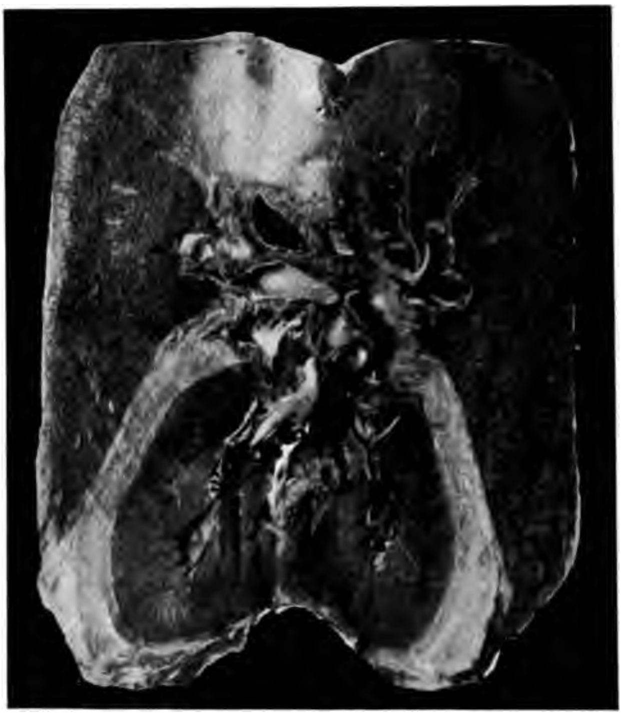

FIG. 381.—Mediastino-pericarditis.

Death is seldom due to the pericarditis itself. It occurs either as the result of degenerative changes in the heart muscle or to exhaustion of the heart as the result of some intercurrent affection.

The relative frequency of the condition and the fact that it is encoun- d far more often at the autopsy table than during life indicate that, ept under unusual circumstances, the lesion is not of great importance.

Symptoms.—There are no symptoms which suggest the presence of erent pericarditis. In what has been designated as the silent group, existence of adhesive pericarditis escapes notice entirely as there are her symptoms nor signs referable to the heart.

In the group in which the ill health is obviously cardiac in origin the gnition of adhesive pericarditis will depend largely on the thorough- s of the physical examination. If it is assumed that the hypertrophy ilatation of the heart is purely cardiac in origin, or that it is secondary vascular or renal disease, the true cause will not be suspected. Fur- rmore, it only too frequently happens that in addition to the adherent icardium there is also an endocarditis. Under these circumstances s usually taken for granted that the endocarditis is the only condition sent. Whether cardiac murmurs are present or not, adhesive peri- ditis is to be thought of in a young adult if there is a history of acute umatic fever followed by endocarditis, and especially so if the cardiac ure is more marked or the cardiac enlargement more extensive than endocardial damage seems to warrant. An additional point of some portance is the fact that in young individuals apparently suffering m endocarditis, an adherent pericardium is to be suspected when the rt does not respond to digitalis.

The recognition of adhesive pericarditis seems to be as uncertain to- y as it was a century ago. Corvisart[1] expressed the diagnostic diffi- ties as follows: "From its insensible attack, from its secret progress, ses in most cases a difficulty often insurmountable in the diagnosis this inflammation. If we attend to the few observations transmitted us on this point of practice, we know not by what signs to distinguish attack, what symptoms accompany its progress; hence we are obliged grant that all the phenomena that belong to this disease are so vague, t it is even uncertain whether a combination of a great number of ervations of this kind can throw much light on its history." Gee has ressed the opinion that in the discovery of adherent pericardium we lom get farther than a guess.

Physical Signs.—*Inspection.*—It is doubtful whether a diagnosis of erent pericarditis can be made unless systolic retraction of the inter- ces is present. The retraction is systolic in time and occurs in the ion of the apex. Systolic retraction also should be looked for poste- ly on the left side between the eleventh and twelfth ribs (Broadbent's n). In extreme cases the costal cartilages or xyphoid cartilage may pulled in with each systole. Epigastric systolic retraction is a normal nomenon and must not be confused with the aforementioned forms.

The character of the cardiac impulse is not of much value as it re- bles that encountered either in hypertrophy or dilatation of the heart. stolic collapse of the cervical veins is cited as an evidence of adherent icarditis, but it is a sign of relatively little value.

Palpation.—The most notable finding on palpation is the presence of stolic shock. This sign ranks with systolic retraction as being the t important evidence of the existence of obliterative pericarditis.

"Essay on Organic Diseases of the Heart and Great Vessels," Eng. Trans. by s, 1812.

The pulsus paradoxus is commonly present, but its diagnostic value is relatively unimportant.

Percussion.—If the pericarditis is also associated with a mediastinitis, percussion may show that the anterior margin of the left lung is immovable and does not change with inspiration and expiration. The normal lateral mobility of the heart upon change of posture, does not occur.

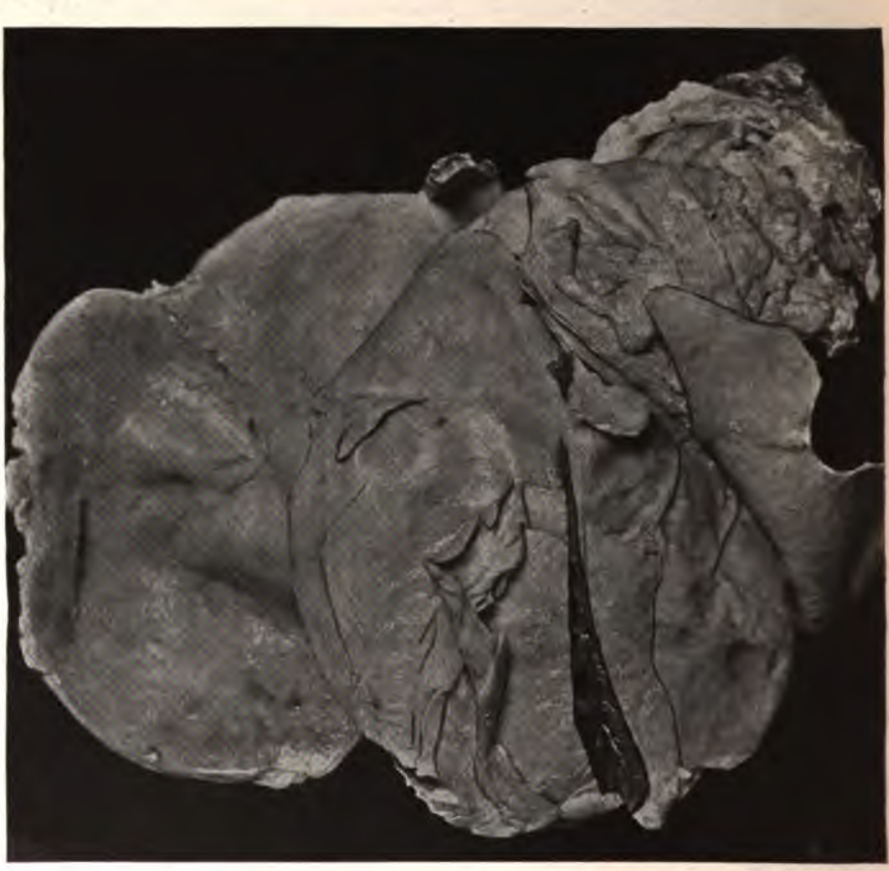

FIG. 382.—Chronic adhesive pericarditis. This specimen illustrates the "Zuckergussherz," so called owing to the resemblance of the exudate to the sugar icing on a cake. It is generally met with in association with "pericardial pseudocirrhosis of the liver." (*Specimen from the Lankenau Hospital, Philadelphia.*)

Auscultation.—There are no auscultatory signs distinctive of adhesive pericarditis. As has been already pointed out endocardial murmurs may be present. If they are, it is to be borne in mind that they do not vitiate a diagnosis of adherent pericardium inasmuch as the association is a frequent one. Among 46 cases in which a presystolic apical murmur occurred without mitral stenosis, Phear[1] found that 20 were associated with adhesive pericarditis. The explanation which has been offered to explain this murmur is that vibrations may be set up by the current propelled from a dilated and hypertrophied auricle into a ventricle whose muscular walls are deficient in tone.

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[1] *Lancet*, 1895, ii, 716.

Auscultation of the lungs may reveal bronchovesicular breathing at the left scapular angle due to compression of the lung.

Pick's Disease or Pericardial Pseudo-cirrhosis of the Liver.—When in addition to the pericardium, the mediastinum, pleura, liver and omentum are also involved, we have what has been described as pericardial pseudo-cirrhosis of the liver, or Pick's disease. The heart and liver are covered with a thick white layer of inflammatory product, giving them the appearance of being coated with an "icing," hence the German name "Zuckerguss Leber and Herz" (Fig. 382). The condition is often tuberculous in origin. Head has collected 55 cases from the literature and added 4. This symptom-complex, sometimes called multiple serositis, presents the clinical picture of cirrhosis of the liver. Ascites and dropsy of the lower extremities are the most prominent signs while the upper part of the body remains free from edema. Jaundice is absent or very transient in character, and aside from the ascites there is no other evidence of portal obstruction. Dyspnea and engorgement of the veins of the neck are usually present in Pick's disease, but the frequency of associated cardiorenal disease in true cirrhosis is apt to produce the same phenomena.

The recognition of this condition will depend, almost entirely on the detection of the signs indicative of adhesive pericarditis. If such signs are present the diagnosis is possible, otherwise the assumption is certain to be that of cirrhosis of the liver as the cause of the trouble.

CHAPTER XXVI

DISEASES OF THE MYOCARDIUM

Introduction.—Valvular disease of the heart is, with few exceptions, easily determined. Changes in the heart muscle, on the other hand, are often extremely difficult to recognize. Although the term "heart disease" is, in the minds of many, associated with a valvular defect it cannot be too strongly emphasized that the ability of the heart to carry on its functions is dependent, almost entirely, on the condition of the cardiac muscle. The majority of valvular defects are for a long time, and, in some instances always, of secondary importance. So long as the cardiac muscle remains healthy, the heart, subject to chronic valvular disease is capable of performing its work as effectively as a normal heart. It is, of course, understood that such a heart is functionally normal under ordinary conditions; it cannot often withstand unusual demands. When, however, the heart muscle becomes exhausted as the result of compensation having reached its limit or when the muscle becomes impaired through degenerative changes, with or without a valvular defect, the most serious circulatory disturbances take place. The diagnosis of structural changes in the heart muscle is often beset with difficulty. Hypertrophy of the heart is, as a rule, easily recognized; so too is the dilatation which succeeds hypertrophy. Degenerative changes, on the other hand, are often entirely overlooked although the etiological factor should, in many instances, put us on our guard. This is particularly true in cases of diphtheria where it is well recognized that acute degenerative changes in the muscle are relatively common and may end fatally unless the possibilities of such a lesion are recognized. Broadbent has stated the case very clearly. "Too commonly no attempt is made to recognize the existence and extent of degeneration or dilatation. The symptoms due to derangement of the circulation force themselves upon the attention of the medical man, but no murmur being detected, the only diagnosis ventured upon is that of 'weak heart,' a vague term which covers the entire ground, from temporary functional debility to disease inevitably and imminently fatal."

The structural changes which may take place in the heart are: (1) hypertrophy, which may be physiological or pathological; (2) dilatation of one or more of the chambers; (3) degenerative changes, which may be acute or chronic; (4) unusual conditions such as syphilitic gummata, abscess and aneurism.

HYPERTROPHY OF THE HEART

The size of the heart is proportionate to the body weight, and to a lesser extent to stature. In an adult man the normal heart averages 300 grams in weight; in an adult woman, 250 grams. As the result of disease the weight of the heart may be enormously increased beyond these limits.

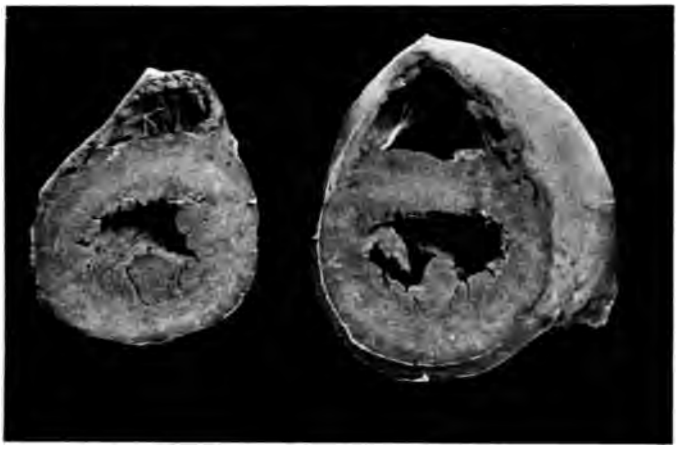


Fig. 383.—Simple hypertrophy. (*Jefferson Medical College Museum.*)

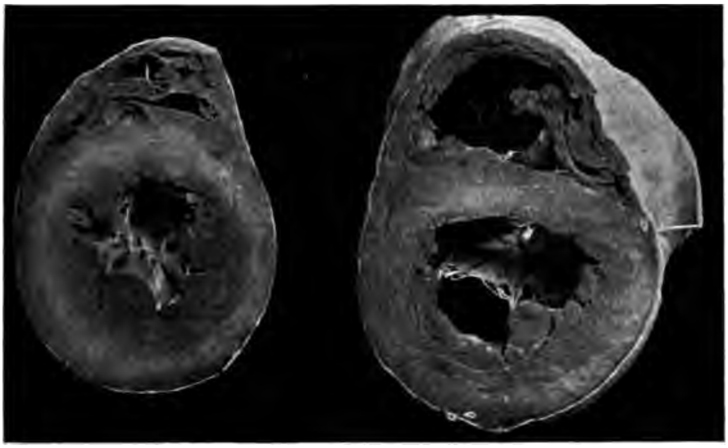


Fig. 384.—Hypertrophy and dilatation. (*Jefferson Medical College Museum.*)

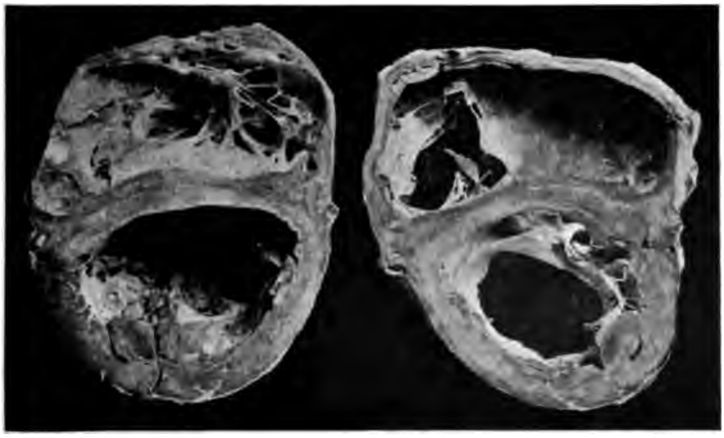


Fig. 385.—Dilatation. (*Jefferson Medical College Museum.*)

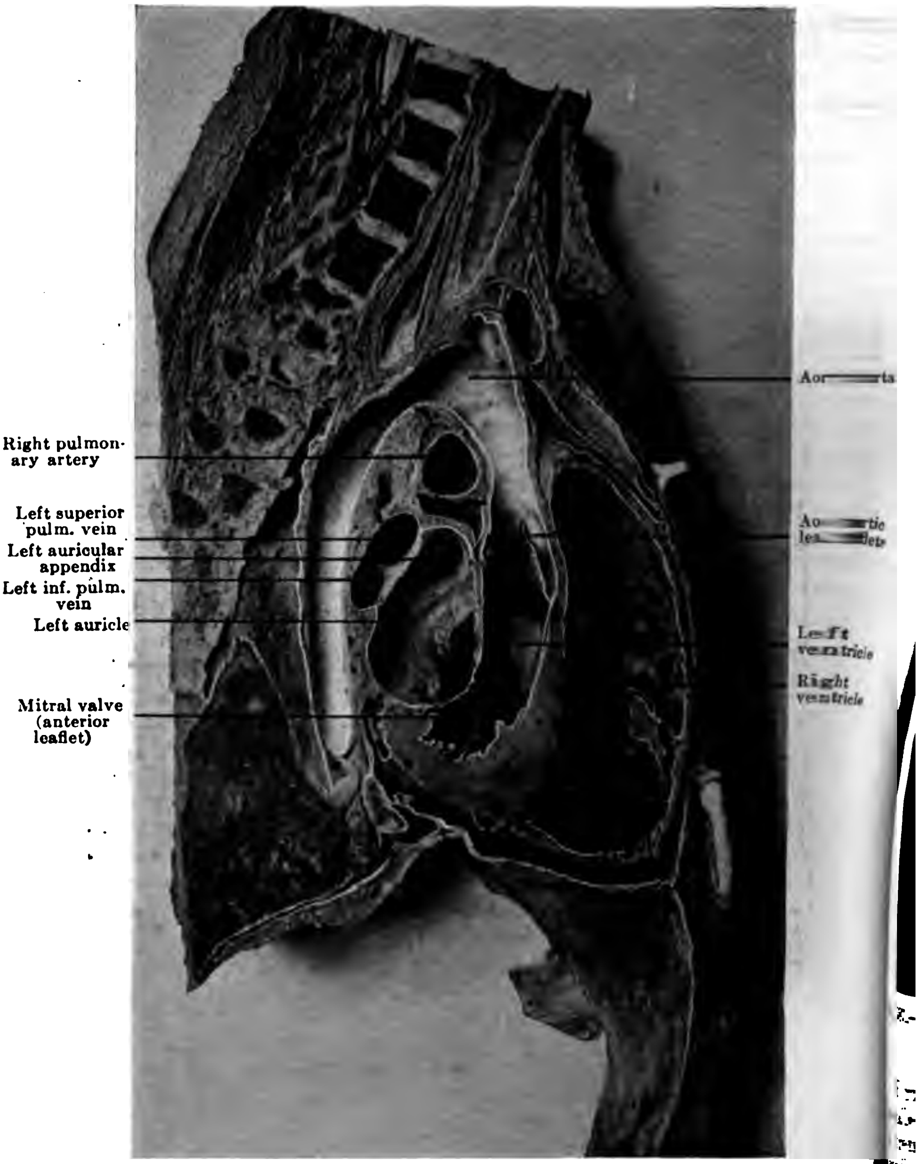
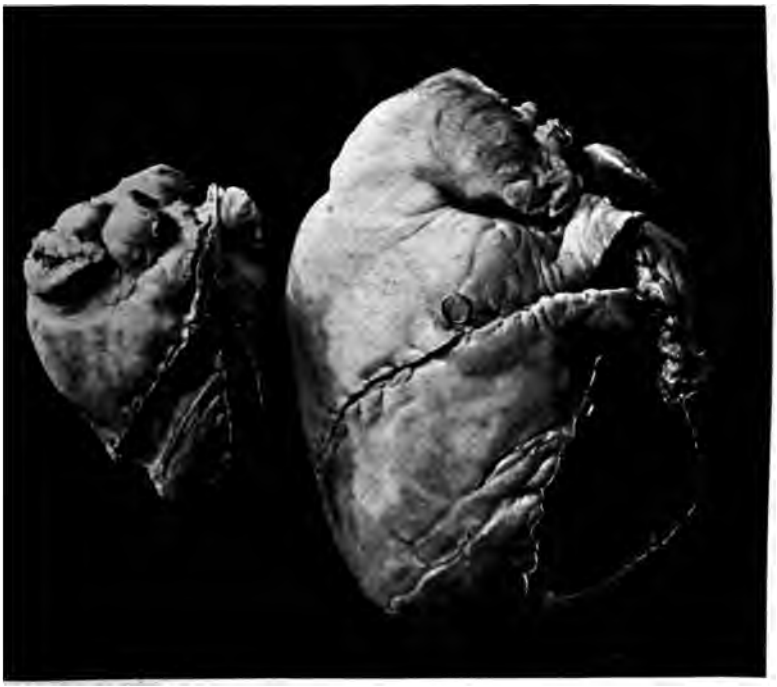


FIG. 386.—Sagittal section of the thorax. Illustrates how little room for expansion there is for the heart between the vertebral column, and the anterior thoracic wall.

ances have been recorded in which the heart weighed 1700 and 2500 s respectively. In men the weight of the heart to the whole body is t 1 to 170; in women it is 1 to 183.

Iypertrophy may affect one chamber only, but this is unusual. The ber to be first affected is that one which first is subjected to an unl amount of work. Generally speaking the left ventricle is the chamn which the hypertrophy is most marked even when other portions of eart are involved also (see Fig. 193).

'wo forms of hypertrophy are recognized: (1) simple enlargement, out change in the size of the cavities; and (2) hypertrophy plus dilan of the cavities, sometimes spoken of as eccentric enlargement. 383, 384, and 385 show very well the change from simple hyperıy to dilatation.



87.—A large hypertrophied heart contrasted with one of normal size. From a case of cardiorenal disease. (*Jefferson Medical College Museum.*)

ormerly another form of hypertrophy was described, namely, the lled concentric enlargement of the heart. In this state the muscle emendously thickened and the cavity diminished in size. This ition is now recognized as a post-mortem change. It is not infretly seen when the autopsy has been held within six hours of death is due to rigor mortis.

Iypertrophy of the heart consists of an increase in either the number e size of the muscle cells, or both. In addition there is also an increase e amount of connective tissue and fat. The hypertrophied muscle is of a brownish-red color and the consistency is markedly increased. Slight degrees of hypertrophy are not apt to cause any disturbance but when the enlargement becomes excessive the mere size of the organ may produce cardiac symptoms. That lack of space may mechanically interfere with the action of the heart is shown in Fig. 386. The most extreme degrees of hypertrophy are those which occur as the result of aortic regurgitation, chronic adherent pericarditis and arterio-sclerosis and chronic nephritis (Fig. 387).

PHYSIOLOGICAL HYPERTROPHY

At birth the thickness of the walls of the two ventricles is the same but as the left ventricle thereafter assumes the chief burden of carrying on the circulation it becomes physiologically hypertrophied to meet the demand. Throughout adult life the heart possesses the ability to increase its muscular power in exactly the same way that the skeletal muscles respond to increased work or exercise. The hypertrophy under these circumstances is, however, rarely of the extreme degree that occurs as the result of pathological conditions.

The normal heart may, therefore, become hypertrophied as the result of laborious occupations or as the result of *athletics*. The effect of competitive athletics upon the heart has received a great deal of attention during the past decade. So far as one can judge from the literature on the subject opinions as to the hurtfulness or harmlessness of competitive athletics are extremely contradictory. That some hypertrophy does occur is unquestioned but as to whether the heart also becomes dilated or whether it later in life becomes more susceptible to endocardial and myocardial changes is still open to question. In a recent study of the effect of rowing upon the young adult heart Lee, Dodd and Young[1] found that there is very little difference in the size of the heart of men who have been rowing for two to four years, and men who have been rowing over ten years. The hearts of these two groups are very slightly larger than the hearts of a younger group who have not as yet participated in serious competitive rowing. These differences seem to be explained by the differences in age and development.

One is probably safe in assuming that if the heart is organically sound excessive work, aside from causing slight physiological hypertrophy will cause no permanent damage. It has always seemed to me that the apparent deleterious effect of athletics in later life is to be ascribed to changed habits. The man who participates in athletic competition, only too frequently, upon leaving college ceases exercising and leads a sedentary life. As a result the cardiac as well as the skeletal muscles, become flabby and weak.

Pregnancy has been cited as one of the causes of physiological hypertrophy but enlargement of the heart in pregnant women is probably more apparent than real. Owing to the high position of the diaphragm in pregnancy the heart is displaced upward and the area of cardiac dulness is increased because the heart is brought closer to the chest wall, and also because it lies more horizontally.

Symptoms and Signs of Physiological Hypertrophy.—Symptoms are usually wanting in physiological hypertrophy except immediately after severe exertion. At this time there may be a sense of throbbing in the vessels of the neck and of the heart itself. Under ordinary conditions there are no untoward symptoms.

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[1] *Boston Med. and Surg. Jour.*, Sept. 30, 1915.

Physical signs are also trivial as the hypertrophy is rarely as marked as that occurring in pathological conditions. Visible displacement of the apex beat is not conspicuous. Percussion usually shows some increase in the cardiac dimensions. On auscultation the first sound is loud and slightly more prolonged than normal and the second sound is not accentuated. When the hypertrophy is slight, such as occurs in athletes, the blood-pressure is normal. Slight degrees of hypertrophy can best be determined by means of the X-rays. The examination is made with the patient standing. The X-ray tube is placed on a level with the mid-portion of the thorax at a distance of 7 feet (teleoroentgenography).

Physiological hypertrophy may be looked upon as dangerous, if the heart is overacting when the individual is at rest. Under the circumstances the cardiac impulse is seen to be heaving or diffuse, the second sound accentuated and the blood-pressure is raised.

HYPERTROPHY AS THE RESULT OF DISEASE

Hypertrophy may occur as the result of disease of the heart itself or as the result of abnormal conditions in other portions of the circulatory apparatus. Hypertrophy under such circumstances is to be looked upon as a defensive action. In order to overcome a valvular defect or the increased arterial resistance the heart hypertrophies in proportion to the increased amount of work demanded of it. Adami and Nicholls classify the causes of hypertrophy as follows:

I. Obstruction to egress of blood.
  (*a*) Endocardial, from stenosis of one of the valves.
  (*b*) Arterial, from diminution of the arterial lumen, sclerosis, contraction of the smaller arteries, etc.
  (*c*) Pericardial, from complete synechia.
II. Increase in the volume of blood to be propelled.
  (*a*) Actual increase in the amount of circulating blood, plethora, Munich beer heart, etc.
  (*b*) From regurgitation, as in mitral and aortic incompetence.
III. Increase in rate of blood flow.
  (*a*) From tachycardia, as in exophthalmic goitre.
  (*b*) As a response to systemic needs, as in the athlete. (This has been considered under physiological hypertrophy.)

In practically all of these conditions the hypertrophy first manifests itself in the left ventricle but as time goes on, whether because of excessive work, or because the exciting cause constantly increases the demand r additional effort, the hypertrophy becomes general. The hyper- phy manifests itself first in the right ventricle as the result of fetal or ngenital heart disease and as the result of certain pulmonary affec- ons (fibrosis of the lungs, emphysema, etc.).

Valvular Disease of the Heart.—This is by far the commonest cause of hypertrophy and has been considered under "Chronic Valvular Disease" (p. 710).

Chronic Adhesive Pericarditis.—As the result of adhesions between the two layers of the pericardium and, often, with the mediastinal tissues, the action of the heart is seriously embarrassed. The additional work then thrown upon the heart often leads to an enormous degree of hypertrophy.

Arterio-sclerosis and Chronic Nephritis.—Next to chronic valvular disease arterio-sclerosis when associated with chronic glomerulo-nephritis is the most frequent cause of hypertrophy. Hypertrophy arising under these conditions has been considered under chronic aortitis or arteriosclerosis of the aorta (see Fig. 181).

"Munich Beer Heart."—Excessive eating and drinking is a common cause of hypertrophy of the heart and occurs with great frequency in those who consume large quantities of beer. A heart which becomes enlarged as the result of excessive beer drinking has been called the "Munich beer heart," although the condition is not peculiar to that city, nor is it confined exclusively to beer. Excessive eating and drinking are apt to be followed by a temporary increase in the activity of the heart, the frequency of the pulse and the tension in the arteries. This is due to the temporary increase in the specific gravity of the blood, stimulation of the heart and blood-vessels by the products of metabolism and also because the excessive ingestion of liquids temporarily increases the total amount of blood (Strümpell). This so-called plethoric condition is usually seen in middle-aged individuals who indulge freely in the pleasures of the table and lead a sedentary life. Many of the cases of idiopathic hypertrophy properly belong under this category.

Tachycardia.—Enlargement of the heart often occurs as the result of increased cardiac action. The most typical example of this form of hypertrophy is seen in Graves' disease. In some instances the rapid heart action and the goitre may constitute the only symptoms of the disease. Neurotic individuals often suffer from tachycardia and slight cardiac hypertrophy.

Symptoms.—Even extreme degrees of hypertrophy may exist for a long time without producing symptoms. This is natural as the hypertrophy develops as a defensive measure and for varying periods of time the circulatory mechanism suffers no inconvenience. Eventually, however, the increased amount of work tends to exhaust the muscle or the demands on it become so great that compensation can no longer be maintained.

Premonitory symptoms are usually present before the heart becomes definitely insufficient. Thus there may be transient attacks of faintness or dizziness, palpitation, or an annoying sense of fluttering of the heart. Shortness of breath or a sense of precordial oppression is common, especially after exertion. In some instances the violent action of the heart may give rise to pain in the region of the heart, throbbing sensations in the head and neck and to pulsatile noises in the ears. Finally, dilatation supervenes and the symptoms and physical signs of failing compensation appear. These differ in no particular from those occurring in chronic valvular disease.

Physical Signs.—*Inspection.*—The apex beat is displaced downward and outward. It may be but slightly removed from its normal position, or, as in the case of marked hypertrophy, it may be in the sixth or seventh interspace and as far to the left as the mid-axillary line. The cardiac pulse is seen as a gentle heave. In very thick-chested individuals ther the apex beat nor the heaving impulse may be visible, even when onsiderable degree of hypertrophy is present.

Palpation.—This serves to confirm the location of the apex beat and o the rather diffuse and heaving character of the impulse. By pal- ion the amount of hypertrophy can be roughly estimated by the ength of the impulse. The cardiac impulse is not only felt to be cible and diffuse but in addition the overlying ribs may be distinctly ed. The forcibleness of the impulse is sometimes better appreciated pressing the fingers into the intercostal spaces.

In those cases in which the hypertrophy is due in large part to high terial tension (chronic nephritis, lead poisoning, gout, etc.) the pulse ll feel full and tense. It usually takes considerable pressure to com- etely obliterate the pulse. In such cases the blood-pressure is always gh.

Percussion.—Unless the hypertrophy is very slight the area of cardiac ulness will be found to be increased downward and to the left. Dulness the right of the sternum is also increased due to the dilatation and ypertrophy of the right ventricle.

Auscultation.—Unless the hypertrophy is due to chronic valvular isease or slight dilatation is also present, no murmurs are heard. The rst sound is loud, low-pitched and more prolonged than normal and gives ie impression of a powerfully contracting muscle. The second sound is sually very distinct at the apex. At the aortic area the second sound iay be accentuated and ringing in character. In such cases there is sually present a chronic aortitis. Reduplication of the first sound at the pex may be the first intimation of oncoming dilatation of the left entricle.

DILATATION OF THE HEART

Just as in the case of hypertrophy one or all of the chambers of the eart may be involved. In mitral stenosis, for instance, distention of the ft auricle may, for a time, be the only evidence of dilatation. As the ick pressure becomes greater and greater the right ventricle also be- mes dilated as does also finally the right auricle. Generally speaking, e use of the term "dilatation of the heart" has reference to dilatation the left ventricle, although, as a rule, the right ventricle is also dilated.

An increase in the capacity of one or more of the chambers of the art may be due to temporary distention or to permanent dilatation. the term *distention* we have in mind a condition in which the chamber larges temporarily as the result of some unusual physiological demand, d when this requirement has ceased the normal condition returns. us in cases of relative tricuspid insufficiency the right ventricle may come distended temporarily as the result of exertion necessitating at muscular and intrathoracic straining. In such cases the valvular fice is stretched as the result of the distention of the ventricle and a kage occurs. In like manner the enlargement of the left ventricle in npensated valvular disease is to be regarded as a physiological disten- n and not a dilatation, even if the condition is permanent and not nporary. The distention is caused by the increased volume of blood left ventricle is forced to handle. In the great majority of cases pertrophy quickly takes place in order to meet the increased work.

When, however, the heart muscle no longer responds to this demand for increased work, the ventricle then becomes pathologically distended or dilated (Fig. 385).

Dilatation is seen in the most characteristic aspects when the heart muscle is the seat of degenerative changes. Thus as the result of fatty or fibroid degeneration the muscle becomes weak, inefficient and unable to meet the normal demands. Inability of the ventricle to properly empty itself quickly leads to dilatation.

That an anatomical defect is not the sole cause of dilatation is evident from the fact that hearts are seen at the autopsy table in which marked degenerative changes have taken place and yet these hearts during life have performed their work fairly satisfactorily and without becoming dilated. Mackenzie believes that the loss of muscular tonicity is the essential feature of dilatation. So long as the tonicity remains unimpaired, or is only slightly diminished, dilatation does not occur even when there is present a serious anatomical lesion. When, however, the tonicity is lost either as the result of the exhaustion of a hypertrophied muscle or because of disease, the heart dilates.

This has been demonstrated experimentally. Thus if extra work is suddenly demanded of the left ventricle as the result of clamping the aorta, the chamber dilates (physiologically distends) and the systolic output is diminished. In healthy hearts there occurs, as the result of increased tonicity, an increase in the systolic output and the ventricle empties itself properly. If, however, the obstruction is too great or the muscle is incapable of responding, the ventricle becomes pathologically dilated (Frank, Hirschfelder).

Dilatation, therefore, may be defined as a condition in which as the result of loss of muscle tone, a diseased heart muscle or one which finally exhausts its reserve power is no longer able to carry on the extra work demanded of it.

Dilatation of the heart may be acute or chronic. Acute dilatation may occur as the result of excessive muscular exertion, especially in individuals out of training or as the result of toxic processes, such as diphtheria, typhoid fever or pneumonia. Sudden mental shock also may lead to an acute dilatation of the heart.

Chronic dilatation is often associated with or is the sequel of hypertrophy. It usually occurs as the result of inadequate nutrition and increased endocardial pressure and to an increased demand for hypertrophy.

Degenerative changes in the heart muscle are very commonly associated with dilatation. At the autopsy table the dilated heart is found to be much enlarged and distended with blood. The walls are thin, flabby, softer than normal and often pale in color from fatty changes. Because of thinness of the wall of the right ventricle this chamber usually shows the most marked evidence of dilatation. The auriculo-ventricular orifices are widely dilated and the septa may bulge toward the less affected side.

Symptoms and Physical Signs of Dilatation.—The symptoms of cardiac dilatation vary greatly. They may consist of nothing more than slight dyspnea and a sense of precordial oppression after exertion. If, however, the ventricle habitually fails to expel all of its contents, the symptoms become more and more marked and the failure of the circulation manifests itself in distant organs and tissues. The subcutaneous tissues become edematous, the lungs congested, effusions take place in the us cavities and the liver becomes engorged. The urine is scanty in unt, of a high specific gravity and contains much albumen.

Often there will be present reflex sensory symptoms as shown by areas yperalgesia of the skin and muscles of the left chest, axillary fold and sternomastoid and trapezius muscles (Mackenzie) (see p. 44).

The physical signs of dilatation are as follows: Displacement of the x beat downward and outward; diffuse pulsation; the evidences of eral venous engorgement; marked increase of cardiac dulness in the sverse diameter; the presence of murmurs of relative insufficiency at mitral and tricuspid areas; and loss of the muscular quality of the sound.

The subject of failing compensation is considered more in detail under heading *chronic valvular disease.*

MYOCARDITIS

The term myocarditis is not altogether a satisfactory one. It is ained because, by common usage, it implies disease of the myocardium. ictly speaking myocarditis is a misnomer inasmuch as many of the ditions embraced under the term, are of a degenerative rather than an inflammatory nature. These changes impair the structure and ctional activity of the cardiac muscle, and may affect all or only a tion of the myocardium. In many instances the ganglia and nerves the heart are also involved. The severity of the lesion will depend in ne cases, on its extent and in others, on its location. The location is extremely important factor for if the muscle fibers composing the car- c conduction path (auriculo-ventricular bundle) are diseased, the ults are much more serious than if an equal amount of non-specialized ue is implicated. It is important to bear this fact in mind as there many cases which during life present marked symptoms of cardiac in- ficiency and yet at the autopsy no evidence of myocarditis is found. e true explanation of this discrepancy probably lies in the fact that a tine autopsy is apt to overlook these small specialized areas which may the only portion of the myocardium involved. Disease of the auriculo- tricular bundle (bundle of His) possesses a pathology of its own and be considered separately (pp. 171 and 196).

Myocarditis may occur in an acute and in a chronic form.

ACUTE MYOCARDITIS

Etiology.—With few exceptions acute myocarditis occurs as the result ome acute infectious process. Involvement of the myocardium is so uent in certain of the acute infections that the possibility of its ence should always be borne in mind.

Acute rheumatic fever is one of the most important of the acute infections his regard. It is not sufficiently appreciated that in most cases of matic endocarditis the myocardium is also involved, and it is to the er that many cases of cardiac hypertrophy and chronic myocarditis their origin. Coombs[1] has pointed out that death in the rheumatic litis of childhood is generally directly due to the myocardial lesions.

Diphtheria.—It is well known that in diphtheria serious myocardial changes are often present and that sudden death is not an infrequent occurrence. The slightest evidence of cardiac weakness or irregularity, other than sinus arrhythmia, during or immediately after an attack of diphtheria is to be looked upon as serious. In such cases every precaution should be taken to avoid even the most trifling exertion.

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[1] *Quarterly Jour. Med.*, October, 1908.

Influenza is often associated with marked myocardial changes. It is important to remember that the evidences of cardiac mischief usually appear some time after the disappearance of the primary disease. In the aged or in the debilitated, sudden death from cardiac failure, several weeks after apparent recovery from influenza, is not an unusual accident.

Syphilis.—It is now recognized that myocardial changes may occur in the early stages of syphilis (see "Myocardial Changes Due to Syphilis").

Typhoid fever is quite constantly associated with myocardial changes. Although sudden death, due to cardiac failure, is rare in typhoid fever, the heart is often permanently damaged to a greater or lesser extent. Acute myocarditis may occur also in *scarlatina*, *smallpox* and *gonorrhea*.

Sudden *blocking of one of the coronary vessels* by an embolism or thrombus is a not infrequent cause of localized acute myocarditis. The sudden shutting off of the blood supply leads to a very rapid degeneration or acute necrosis in the affected area. If the embolus is septic in origin an abscess results. Acute abscess formation in the myocardium may be part of a general pyemic process such as occurs in puerperal fever or malignant endocarditis. Among 9940 autopsy records analyzed by Norris there were 6 cases of *acute suppurative myocarditis*.

Morbid Anatomy.—The pathological picture of acute myocarditis will depend on the exciting cause. The most frequent of the myocardial changes is an acute parenchymatous degeneration which results from the action of toxins in the course of the acute infections. It is a diffuse process and varies greatly in severity. It may consist of nothing more than a slight cloudy swelling. In well-marked cases there is in addition a granular degeneration of the muscle fibers. The myocardium then presents a pale turbid or opaque appearance which has been likened, by Adami, to parboiled meat or raw fish. The muscle is soft, flabby and readily torn. The ventricles, especially the left, are usually dilated. Microscopically the muscle fibers are seen to be swollen, fragmented and the striations more or less obscured by minute granules which are probably albuminoid in nature.

Less frequently an acute interstitial myocarditis occurs as the result of one of the acute infections or acute pericarditis. In this form there are collections of inflammatory leukocytes between the muscle fibers and connective-tissue proliferation. Minute hemorrhagic areas are often present also.

When the change has been slight in degree, recovery of the muscle is usually complete. In other instances the damage is so great that death ensues as the result of cardiac failure; in still other instances the muscle remains permanently impaired and a chronic myocarditis eventually develops.

When the nutrition of the heart is interfered with as the result of the sudden blocking of one of the branches of the coronary artery a localized myocarditis occurs. This may be septic or benign in character. The former is encountered in cases of general sepsis in which the finer branches of the coronary artery are blocked with septic emboli. As a result small scesses develop in the myocardium. These vary in size from a pinad to a split pea.

The benign form may occur as the result of an embolism or thrombosis a coronary artery, the seat of atheromatous changes. In such cases ɜ blood supply to a given area of the myocardium is suddenly shut . It is variously designated anemic necrosis, acute softening and /omalacia cordis. Cardiac infarcts are most commonly seen in the t ventricle and septum which are supplied by the anterior coronary tery. The appearance of the infarct depends on its age. The changes ıich follow blocking of one of the branches of the coronary artery are ually rapid. At first the affected area is yellow or grayish-red in color. is of an irregular or wedge shape and projects slightly above the surce. The initial change is a coagulative necrosis and softening. The eakened point in the heart wall may rupture and cause sudden death. his is rare. The usual termination is the transformation of the infarct to scar tissue.

Symptoms.—It too often happens that the physician in his anxiety 'er the symptoms due to the primary disease loses sight of the secondary anges which may occur. At the best, the initial symptoms indica'e of myocardial weakness in the acute infections are slight, easily erlooked and may be entirely absent. The sudden death of the patient ıy be the first intimation that cardiac weakness has been present. In ne instances the myocardial weakness manifests itself during the rile stage; in others, notably influenza and diphtheria, circulatory turbances may not appear until much later.

The symptoms of myocardial weakness are restlessness or apathy, eathlessness on exertion, a sense of constriction in the chest and, at ıes, pain which is anginoid in character. In the more serious cases ere will be evidences of dilatation of the heart, namely, cyanosis, spnea, precordial and hepatic pain, vomiting and edema.

Physical Signs.—*Inspection.*—In the severe cases the patient often esents a pallid appearance and is either apathetic or very restless. If atation has occurred there may be cyanosis and more or less edema of e lower extremities. Inspection of the precordium may be negative it if considerable dilatation has taken place the apex beat is displaced the left and the impulse may be diffuse.

Palpation.—This serves to locate the apex beat and to determine the rce of the impulse. Of more importance is the character of the pulse. he most common change in the pulse is feebleness with or without an icrease in the rate. Much less frequently there is a *bradycardia.* Unue slowness of the pulse is most often encountered some weeks after covery from an attack of influenza. The bradycardia may be vagal origin or it may be caused by changes in the auriculo-ventricular bundle. he most serious change to be noted in the pulse is lability and arrhythia. The latter may be due to changes in the force of the respective ats or to alterations in the rhythm. The blood-pressure in acute yocarditis is usually low.

Percussion.—Unless considerable dilatation is present there will be change noted. An increase in the transverse diameter of the heart eaks for dilatation.

Auscultation.—If the myocardial weakness is at all marked the heart unds are accentuated and short and the first sound lacks the normal muscular quality. The second pulmonic sound is usually sharply accentuated. Either the first or second sound may be reduplicated; if reduplication of the former occurs it may be the first intimation of dilatation. A functional murmur at the mitral area is common and in some instances a similar murmur may be heard also at the tricuspid area.

Diagnosis.—As has been pointed out, the diagnosis of acute myocarditis is not easy and in many instances is a matter of conjecture. Armed with the knowledge that the condition is a frequent accompaniment of the acute infections, the physician should always be alert to detect the slightest alterations in the circulatory mechanism. No matter how trivial these may appear, they should be given serious consideration and measures taken to guard the patient against undue exertion. It is important to bear in mind that the evidences of myocardial weakness may not appear until several weeks after the patient has recovered from the acute infection. For this reason patients who have recovered from influenza and diphtheria, especially the latter, should be kept under supervision.

CHRONIC MYOCARDITIS

Etiology.—The causes of chronic myocarditis are extremely varied. In every instance the chronic lesion is probably the sequel of an acute process but in the great majority of instances the latter passes unrecognized. As a rule the progress of the myocardial change from an acute inflammatory or degenerative process to one of a chronic nature is insidious and gives no evidence of its presence until more or less serious muscular insufficiency develops. Not infrequently urgent cardiac symptoms develop suddenly and in the midst of apparent health. If this occurs in relatively young or middle-aged individuals syphilis is to be suspected.

One of the most important causes of chronic myocardial changes is disturbance of the coronary circulation which results in poor cardiac nutrition. This may be abrupt as the result of an embolus or thrombus in which case a coagulative necrosis takes place and later scar tissue is formed. More commonly the nutrition of the heart muscle is lowered by reason of sclerosis of the coronary vessels. The extent of the myocardial changes will depend on the amount of vascular damage. The causes of the coronary sclerosis are the same as those producing arteriosclerosis elsewhere. The blood supply to the heart may be seriously interfered with by closure of the mouths of the coronary vessels as the result of disease of the first part of the aorta, the coronary vessels themselves being healthy. This is especially apt to occur as the result of *syphilitic aortitis*.

It is well known that the final stage in chronic valvular disease is, in the great majority of instances, muscular insufficiency due to myocardial degeneration. This is brought about partly by increased work and p
by perversions in cardiac metabolism. Similarly the hypertroph
dilatation which result from persistent cardiac strain whether d
physical exertion, to hypertension in the arteries or to adherent peric
tis, lead to myocardial degeneration because of the increased work
manded of the heart. There may be included in this group also th
instances in which the heart for long periods of time beats at a grea
increased rate, the most notable example of which is seen in exophthalm
goitre.

Myocarditis has also been attributed to high-pressure methods of living, severe physical labor and hardship and the chronic infections, especially syphilis. Inasmuch as these factors are very frequently the underlying causes of general arterio-sclerosis it is probable that sclerosis of the coronary vessels is the actual cause of the myocardial changes.

Finally degenerative changes may be brought about by chronic intoxications, such as perversions of metabolism, alcoholism, nicotine poisoning, phosphorus poisoning, the prolonged use of arsenic, etc.

Christian[1] in a study of 407 cases of chronic myocarditis found that none of the commonly given etiological factors was entirely satisfactory and that in some instances there was no adequate explanation as to why the muscle failed.

The muscular insufficiency usually manifests itself first in that portion of the heart in which the strain has been greatest. Thus in general arterio-sclerosis, aortic insufficiency, and the hypertrophy due to excessive exertion, it is the left ventricle which is subjected to the greatest strain. On the other hand, chronic pulmonary lesions, such as emphysema, asthma or fibrosis of the lungs and mitral lesions, throw the greatest amount of work on the right ventricle and in such cases it is this chamber which is the first to become embarrassed.

Morbid Anatomy.—The demonstrable anatomical lesions are either of a fibroid or fatty nature. In not a few instances, however, neither the gross nor the microscopic examination reveals the cause of the failure of the muscle although the clinical picture in these cases is identical with those showing definite changes.

Fibrous myocarditis or fibroid degeneration may be limited to a small localized area or there may be numerous small patches of fibrosis or the change may be diffuse in character. The localized patch following an infarct from blocking of one of the coronary vessels has been referred under acute myocarditis. The patchy and diffuse forms are most frequently due to sclerosis of the coronary arteries. In some cases numerous small areas of anemic necrosis undergo fibroid changes; in others, the interference with the blood supply causes a slow and gradual fibroid degeneration. Fibroid areas may result also from areas of acute inflammation or as the result of the action of a toxin.

The fibrosis is found most often in the apex of the left ventricle and in the septum (Fig. 388). It may be limited, however, to the papillary muscles, the right ventricle, or the auricles. Aneurism of the heart wall always has its origin at the site of a fibroid area. Rupture of the heart is an occasional accident resulting from chronic fibrous myocarditis.

Fatty Heart.—This is a relatively common condition. Two forms are recognized, namely fatty degeneration and fatty infiltration or fatty overgrowth.

Fatty degeneration is met with in cachectic conditions, in prolonged infections, and as an accompaniment of or sequel of acute parenchymatous degeneration. The condition often occurs in an extreme form in pernicious anemia and phosphorus poisoning. Disease of the coronaries may cause fatty degeneration although fibroid changes more frequently result.

The heart muscle is pale in color, and soft in consistency, so that the finger readily penetrates it. The heart may be so softened and so flabby that it entirely loses its normal contour when removed from the body.

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[1] *Jour. Amer. Med. Assoc.*, June 22, 1918.

Very commonly the muscle will show streaks or patches of yellowish-brown, the so-called "tabby cat" striation. The striations are usually well marked in the papillary muscles.

Fatty degeneration is usually most apparent in the left ventricle, near the apex. The right ventricle, the septum and auricles are involved in the order mentioned.

Microscopically tiny globules of fat are seen deposited in the muscle fibers and not between them.

Fatty infiltration or fatty overgrowth is a condition in which the heart is encased in a thick layer of fat. In extreme cases the muscle may be completely hidden. In addition fat is deposited between but not in the muscle fibers. Usually fatty infiltration is a part of general obesity but it occasionally is met with in the absence of undue stoutness.

Brown atrophy is a common condition and is usually found in association with valvular lesions and in senile hearts. The color of the heart muscle is reddish-brown and the consistency is much increased.

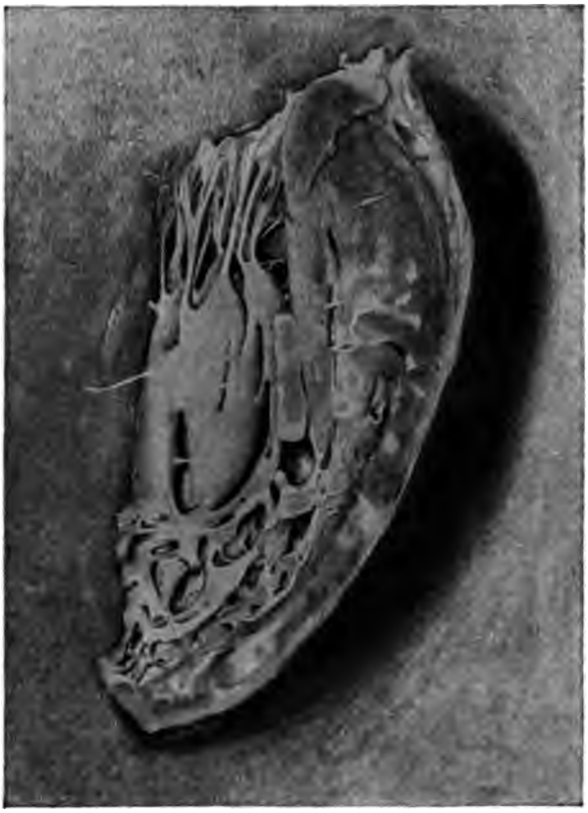
Of the more unusual anatomical lesions may be mentioned fragmentation and segmentation of the muscle fibers, calcareous degeneration, amyloid degeneration and hyaline transformation.

Inasmuch as the physical signs of mitral regurgitation are usually present the autopsy records of the Peter Bent Brigham Hospital are of interest, in that they show that the mitral valve was normal in 32 out of 41 cases (Christian).

Symptoms.—The clinical picture of chronic myocarditis is a varied one. Not infrequently symptoms are absent or very trivial in character and yet well-marked lesions are found at the autopsy. Sudden death may be the first intimation that a serious cardiac lesion is present. On the other hand, the clinical picture may point clearly to insufficiency of the heart muscle and yet at the autopsy no lesion is found. Cabot in a comparison of the clinical and pathological diagnoses of myocarditis found that the condition was correctly recognized in 22 per cent., overlooked in 26 per cent., and diagnosticated when not present in 52 per cent. Of the last-mentioned group it may be said that small localized areas of disease, particularly in the specialized tissue of the conduction path, may have escaped notice. Careful microscopic studies are much needed in that group of cases which give symptoms of cardiac insufficiency but which at death show no gross anatomical lesion.

Unless symptoms are entirely wanting the one outstanding feature of chronic myocardial degeneration is cardiac insufficiency. To a greater or lesser extent the symptoms which characterize this condition will always be present. Very often, however, the symptoms of cardiac weakness are overshadowed by some special symptom complex, as for instance, angina pectoris, Stokes-Adams disease, or hypertension with or without chronic nephritis.

The first intimation of myocardial weakness may be transient attacks of dizziness or faintness and this is especially apt to occur in robust, middle-aged men with arterio-sclerosis. Breathlessness and a sense of oppression in the chest after some slight but unusual exertion are early symptoms. Pain is especially apt to occur if the exertion follows a meal. The pain passes off with the eructation of gas. In such cases the trouble may be attributed, erroneously, to dyspepsia. Slight swelling of the feet and ankles may be an early manifestation. Associated with the



388.—Old scars in the heart-wall (chronic fibrous myocarditis). (MacCallum, "Text-Book of Pathology.")

dyspnea there may be anginoid attacks of pain and as has been stated, true angina may dominate the picture. In other instances, the patient suffers from collapse in which he sweats and has a feeble but slow pulse.

The symptoms just given may be transient but, as a rule, they tend to become increasingly severe until the characteristic picture of dilatation or incompetence appears. The dyspnea persists, even at rest, and Cheyne-Stokes breathing may occur. Cough is often annoying and the sputum hemorrhagic as the result of pulmonary infarcts. The patient is pale, usually cyanosed and there is often edema of the lower extremities. Owing to the back pressure in the veins the liver becomes engorged and painful and in addition there is nausea and vomiting. In the terminal stages mental symptoms are common.

In obese individuals with fatty overgrowth of the heart, dyspneic attacks, resembling asthma, are common. The puffing respiration of such individuals is usually ascribed to their obesity but more often it is an evidence of cardiac weakness. In some individuals, dyspnea without cyanosis results from *acidosis*, due either to insufficient renal elimination or to failing compensation which causes carbon dioxid retention in the blood. When acidosis as indicated by the alveolar carbon dioxid tension, occurs in cardiac and cardio-renal disease, it is associated with an increased stimulability to carbon dioxid in the inspired air. In addition, these patients are subject to attacks of bronchitis, and to attacks of dizziness and faintness. In some corpulent individuals the face has a dusky, congested appearance which is commonly ascribed to ruddy health but which in reality is an evidence of venous stasis.

Physical Signs.—In taking up the physical signs of myocarditis no mention will be made of the secondary changes which take place in the heart as the result of hypertrophy whether due to valvular lesions or to hypertension. These have been considered under their appropriate headings. The signs described here are those which occur in association with fibrosis of the heart muscle and in the so-called fatty heart.

Inspection.—The apex beat may be seen in its normal position, but as a rule, it is displaced to a greater or lesser distance beyond the nipple line and the impulse is feeble and diffuse. Pulsation of the vessels in the neck may be marked.

Palpation.—The pulse shows the greatest variations. It may be irregular, either as to force or rhythm, and rapid or slow. In many cases it is simply weak. Irregularity is perhaps the most common abnormality but is often not present until the late stages of the disease. The arrhythmia may be due to extrasystoles or to auricular fibrillation. A very slow pulse is not infrequent and often there are associated with it other symptoms characteristic of the Stokes-Adams syndrome. Slight physical exertion markedly accelerates the pulse rate which remains high for some time afterward.

The blood-pressure varies according to the underlying cause of the myocarditis. In fatty degeneration it is often low but in spite of well-marked cardiac lesions the normal pressure may be maintained or even increased. In the myocarditis of old age the exciting cause is, as a rule, sclerosis of the coronary vessels. Being a part of general arterio-sclerosis, the blood-pressure is high. In obese individuals with fatty overgrowth of the heart, the blood-pressure is usually high.

Percussion.—The cardiac outline is usually enlarged to the left and if dilatation is present, cardiac dulness will be increased.

Auscultation.—The heart sounds are not characteristic: they may be distant and feeble, gallop rhythm may be present, the first sound may be short and sharp and the second pulmonic or second aortic may be accentuated. A systolic murmur is usually present at the apex due to relative insufficiency of the mitral valve. It may be soft and blowing or harsh or have a musical quality and may be transmitted toward the axilla. It tends to disappear with rest and appropriate treatment. On the other hand, it is often possible to bring out such a murmur by having the patient ascend a flight of stairs or walk up and down the room for a few minutes. A murmur developing under these circumstances is practically always an evidence of muscular insufficiency. Because of this murmur and the signs of failing compensation the condition is often erroneously considered to be mitral regurgitation. There may be a tricuspid murmur and a positive venous pulse due to relative insufficiency and, in not a few cases, there is a systolic murmur at the aortic area due to sclerosis or dilatation of the first part of the aorta.

Diagnosis.—Patients in whom a diagnosis of chronic myocarditis seems to be justified are, as a rule, individuals past the middle period of life who develop symptoms characteristic of cardiac insufficiency. On examination the transverse diameter of cardiac dulness is increased; the first sound is feeble and valvular in character and associated with it there is usually a systolic murmur. The second aortic or pulmonic sound may be accentuated and ringing.

The type of case which too commonly escapes recognition is that in which symptoms are absent or too trivial to demand attention. This is the type in which sudden death is often the first intimation that serious cardiac disease is present. Babcock states that myocardial damage may be strongly suspected in individuals of or beyond middle age who have dissipated, overindulged themselves at the table, or lived at high pressure. They will often be found to have a high blood-pressure, arteries which are visibly and palpably thickened and albumen and casts in the urine. Examination of the heart will show some increase in dulness to the left, an intensely accentuated second aortic sound, and accentuation of and a valvular quality of the first sound at the apex. A harsh systolic murmur at the aortic area is a frequent finding (see p. 742).

It is well to bear in mind that the etiology and symptomatology of chronic degeneration of the myocardium and the blood-vessels are often essentially the same and that the presence of one almost invariably implies the other.

ANEURISM OF THE HEART

Aneurism of the heart wall may occur in an acute and a chronic form. The acute type is a sequel to ulcerative endocarditis and usually terminates by perforating into the pericardial sac. It is of pathological interest only.

Etiology.—The condition is rare. In a study of 9940 autopsy records at the Pennsylvania and Philadelphia General Hospitals, Norris found 8 cases. In 1908 M'Elroy[1] found there had been recorded in medical literature 300 cases. Cardiac aneurism is practically always a sequel of chronic fibroid myocarditis.

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[1] *Jour. Am. Med. Assoc.*, Aug. 1, 1908.

Morbid Anatomy.—The condition is usually single although occasionally more than one has been noted. The aneurism is nearly always located in the anterior part of the left ventricle near the apex and usually consists of a globular distention in this region (Fig. 389). At the point of bulging the heart wall is greatly thinned and fibrous in character. The ventricle elsewhere is usually hypertrophied. The endocardium lining the aneurism is often thickened, opaque and may show calcareous plaques. The aneurismal sac may be filled with a thrombus. If the opening into the ventricle is narrow a thrombus usually forms and this tends to relieve the strain on the thin and weakened wall. In the ma-

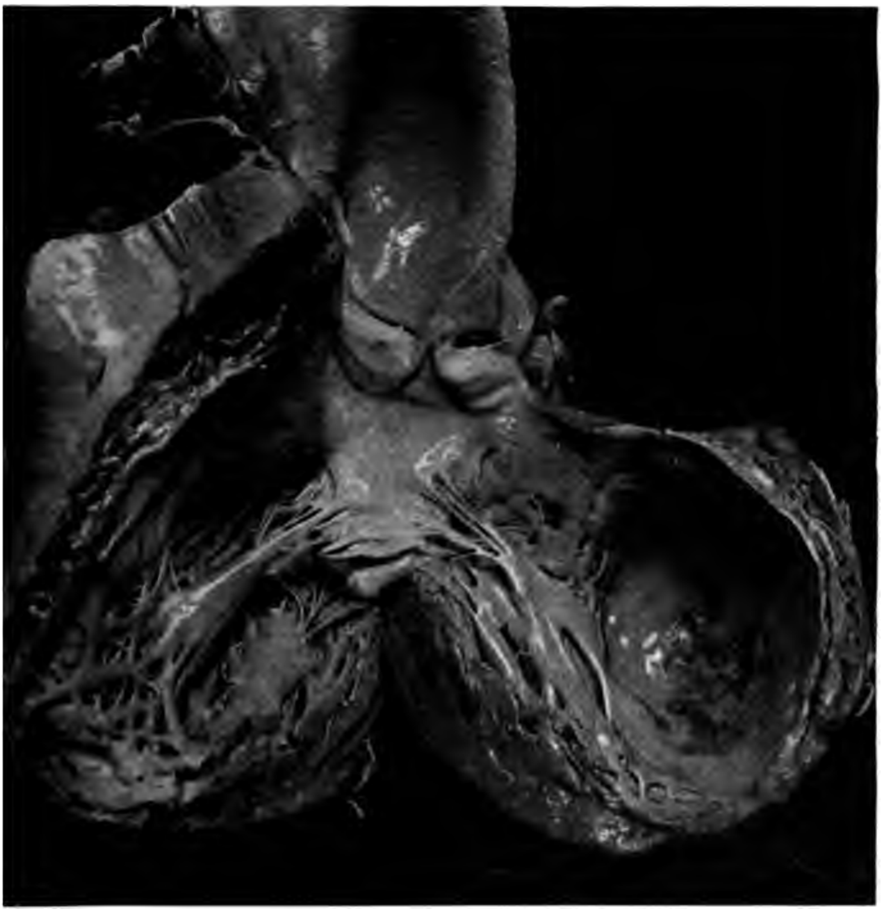

FIG. 389.—Aneurism of the left ventricle. The left ventricular wall, which is elsewhere hypertrophied, is markedly attenuated on its left side at and above the apex. In this region a marked bulging, about the size and shape of a large duck egg, is seen. The endocardium in this region is covered with calcareous plaques. The aortic and mitral valves show moderate sclerosis. (*Specimen from the Philadelphia Hospital.*)

jority of cases, however, the strain proves too great and rupture ta place. The size of the aneurism varies greatly; it may be the size o fist or even larger.

Symptoms and Signs.—There are no symptoms which can be tributed to this lesion. Inasmuch as it is one of the phases of chro fibrous myocarditis the symptoms presented will be those occurring in latter condition. If it is to be recognized at all it will be by the prese of a pulsatile tumor situated at a point beyond the limits of the aorta.

At first sight the condition with which a pulsatile tumor, due t cardiac aneurism, is most apt to be confused is a pulsating empye The absence of signs pointing to fluid in the pleural sac and failure obtain pus with an exploring needle serves to rule out empye Owing to the enlargement of the heart and the change in its contou cardiac aneurism may simulate a pericardial effusion and aspiration performed.

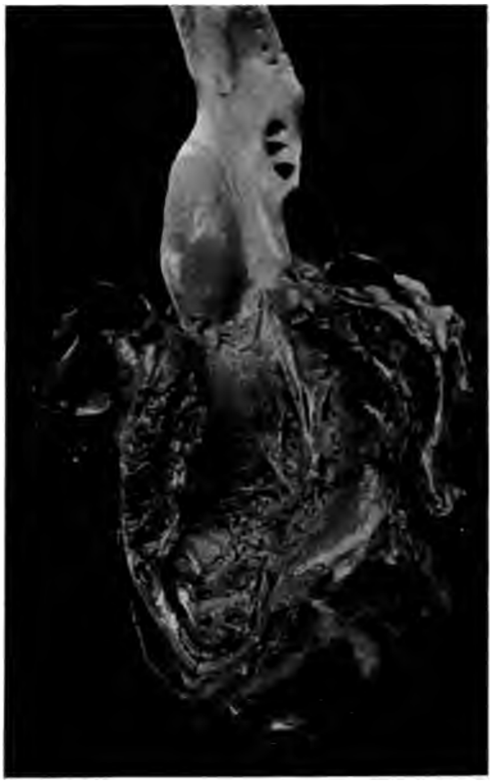

Fig. 390.—Aneurism of left ventricle. (*Patient of Dr. J. N. Henry.*)

The following case was seen at the Pennsylvania Hospital on the se ice of Dr. J. Norman Henry.

The patient, a colored woman, aged forty-two years, was admitted to the hos complaining of extreme shortness of breath, weakness, cough and swelling of the Her husband and several brothers and sisters had died of tuberculosis. For the three years she had had a cough and expectoration and occasional night sweats. She had been married eight years but had had no children or miscarriages. Her present illness began suddenly with dyspnea, palpitation and edema of the legs, a month prior to her admission to the hospital. Her cough had become worse and for two months she had been hoarse.

Physical Examination.—On inspection there was seen in the left axillary region, at the level of the eighth rib, a pulsating tumor which was definitely expansile (see Fig. 14). The heart was displaced to the right. No murmurs were heard. Over the lower half of the left chest the percussion note was flat, the breath sounds were barely audible and vocal fremitus was diminished. The Wassermann reaction was strongly positive. Fluoroscopic examination showed the presence of fluid in the left chest and distinct pulsation. The heart and trachea were much displaced to the right. Later a slight effusion was present on the right side also. The left side was tapped and 900 c.c. of serous fluid withdrawn.

The diagnosis seemed at first to lie between a pulsating empyema and an aneurism. The former was ruled out after the paracentesis as the pulsation persisted. A diagnosis of aneurism of the thoracic aorta was untenable because of the location of the pulsating tumor.

The patient died six months after admission as the result of a cerebral hemorrhage. The heart (Fig. 390) showed the presence of a large aneurism in the left ventricle just posterior to the apex of the heart. The wall of the aneurism was composed of fibrous tissue.

MYOCARDIAL CHANGES DUE TO SYPHILIS

Until comparatively recently the only lesion of the heart that could unmistakably be attributed to syphilis was the gumma, although it has always been recognized that syphilitic subjects were prone to suffer from degenerative changes of the heart muscle. These changes, however, were looked upon as part of a general or coronary arterio-sclerosis. Since the discovery of the spirocheta pallida, histological studies have demonstrated that this organism shows an especial predilection for the aorta and heart. It is now clearly proved that the root of the aorta is especially vulnerable and that in many instances the aortic valves are also implicated. Furthermore, the studies of Warthin[1] and others have shown that the heart muscle itself may be invaded by the spirochetæ and that the infection may occur in the early stages of syphilis. In 41 cases of syphilis studied by Warthin, active syphilitic lesions were found in the heart muscle in 36. Heretofore it has been taught that syphilitic changes in the heart occurred only as late manifestations of the disease.

Morbid Anatomy.—There seems to be a selective tendency on the part of the spirochetæ to attack the coronary vessels. In a series of 50 cases studied by Brooks[2] the coronary arteries in 35 showed a relatively severer degree of change than the remainder of the arterial system. The early syphilitic changes in the heart have been studied by Brooks,[3] Adler[4] and Warthin.[5] The change occurs early in the course of the infection and involves the terminals of the coronary system. The first alteration consists of an infiltration of mononuclear cells in the muscle about the vessels and in addition there are numerous spirochetæ. This is followed by cellular fibrosis and finally necrosis. Brooks urges the importance of bearing in mind the character of the lesion and the fact that it may manifest itself during the so-called secondary period. Vigorously treated, such lesions may be recovered from without permanent damage being done to the heart. Neglected, there may eventually result areas of fibrosis or the formation of gummata.

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[1] *Trans. Assoc. Am. Phys.*, 1914.

[2] *International Clinics*, Twenty-fifth Series, vol. i, 1915.

[3] *Loc. cit.*

[4] *Trans. Assoc. Am. Phys.*, 1898.

[5] *Ibid.*, 1914.

The late cardiac changes due to syphilis are fairly common. In an analysis of the cardiac lesions found in 50 consecutive cases of syphilis Brooks found that the changes in the heart muscle were by far the most common. Fatty degeneration was found in 44; fibroid changes occurred in 4 and in 5 there were fibroid changes and fatty degeneration. Gummata were present in 5 of the 50 cases. Mraček,[1] among 60 cases, found gummata in 10. In common with degenerative changes due to othe causes syphilitic involvement may include or be limited to the specialize fibers, known as the auriculo-ventricular bundle. There are now o record a number of cases of the Adams-Stokes syndrome in which th bundle of His was the site of a gummatous infiltration. An excellen example of this is shown in Fig. 391. The lesions are chiefly encountere

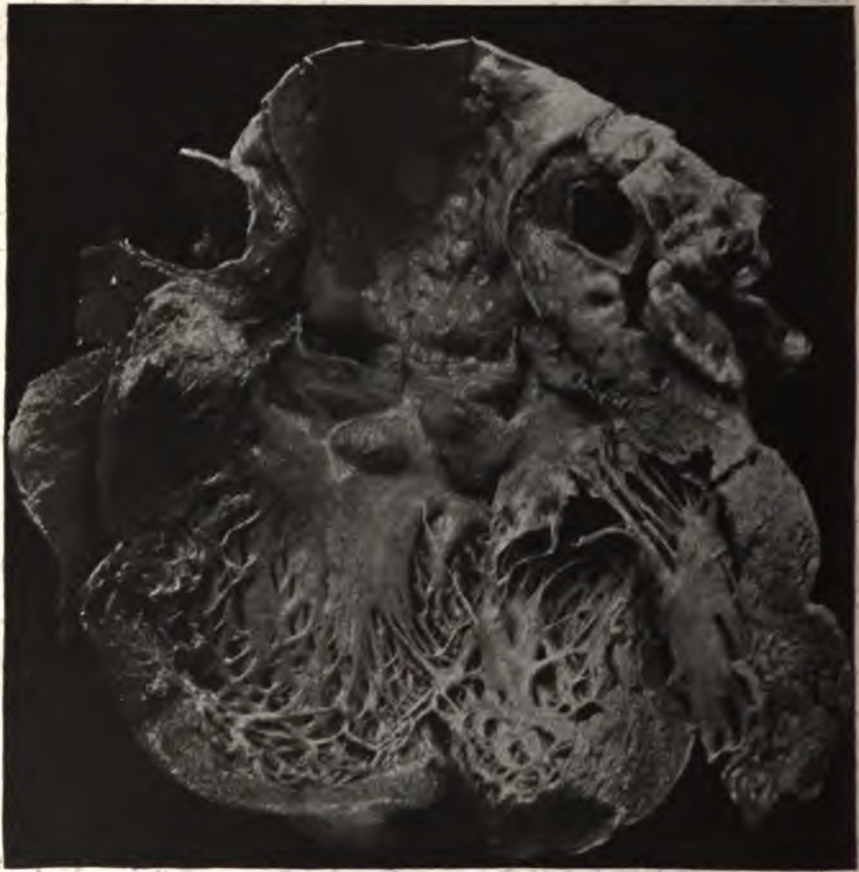

FIG. 391.—The nodular gummatous mass obliterates the undefended space of the interventricular septum, and entirely fills that part of the septum through which the auriculo-ventricular bundle runs. (*Specimen from the Pennsylvania Hospital.*)

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[1] *Arch. f. Dermatol. und Syph.*, 1893, xxv, p. 279.

n the ventricles, are generally multiple and, as a rule, are sharply defined nd encapsulated (see Fig. 392). Absorption of the gumma may be ollowed by cardiac fibrosis or aneurism.

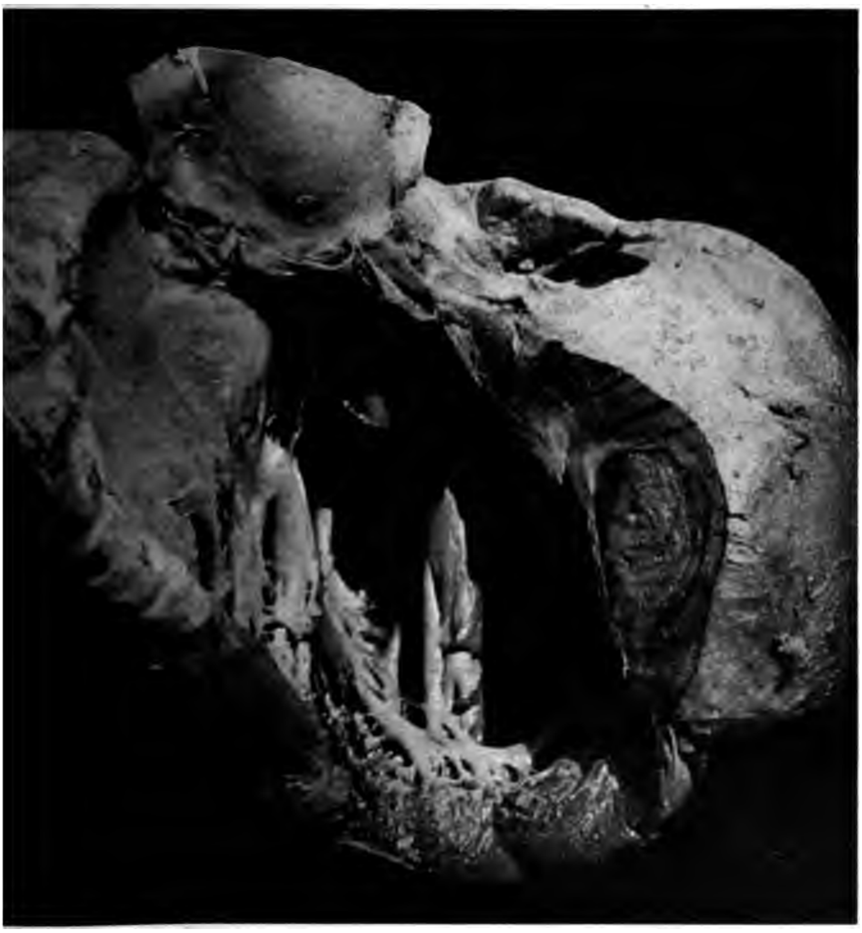

FIG. 392.—Gumma of the left ventricle. (*Specimen from the Pennsylvania Hospital.*)

Symptoms.—The symptoms of the late cardiac manifestations of yphilis differ in no particular from degenerative changes due to other uses. It is not generally appreciated, however, that the heart may be fected early in the infection and that at this time the evidences of severe rdiac weakness may be present. Brooks found that dyspnea was the ost common symptom in the early cases. Precordial pain and tenderss are also common and in some instances anginal attacks are frequent. rassmann[1] also has directed attention to the cardiac symptoms arising early syphilis. He found that dilatation of the right heart was comon and that a functional or accidental murmur at the apex was present in 40 per cent. of his cases. The murmur is soft and blowing in character, becomes more evident on exercise and is occasionally transmitted toward the axilla. In addition, alterations in the cardiac rhythm are almost universally present in the form of arrhythmia, bradycardia or tachycardia. The cardiac involvement may be severe enough to cause death at this time; on the other hand, vigorous antiluetic treatment usually causes a disappearance of the symptoms.

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[1] *Münch. med. Woch.*, 1897, xliv; *Deut. Arch. f. Klin. Med.*, 1900, lxix, 58, 264.

As a rule, the symptoms of the late cardiac manifestations of syphilis differ in no particular from degenerative changes due to other causes. Sears[1] believes that syphilis should be suspected, however, in apparently healthy patients who as the result of overwork or some sudden exertion, suddenly develop urgent cardiac symptoms. These symptoms may consist of tachycardia accompanied by great anxiety and mental distress or by the usual signs of myocardial insufficiency. Severe headache and vomiting may be early symptoms. A striking feature of these cases is their failure to respond to the ordinary cardiac tonics.

Breitmann[2] believes that many supposedly rheumatic heart affections are in reality syphilitic in origin. This should be suggested by the rapid onset of cardiac symptoms, with or without fever, in the midst of apparent health. Death may occur suddenly or the cardiac weakness may persist for a long time without signs of valvular mischief and without being influenced by the ordinary heart tonics.

Diagnosis.—It is to be borne in mind that syphilis may attack the heart early in the course of the infection and for this reason the heart should be carefully examined. The occurrence of cardiac symptoms or signs at this time demand active antiluetic treatment. The recognition of cardiac syphilis as a late manifestation is dependent on establishing the presence of an acquired or congenital infection. In some instances it is frankly acknowledged that such an infection has occurred; in others it is denied. In the absence of syphilitic stigmata the Wassermann test is our main reliance. Syphilis is to be suspected if cardiac symptoms suddenly develop in young or middle-aged individuals who have been healthy previously.

GOITRE HEART

Enlargement of the heart, independent of any valvular, pericardial, arterial, pulmonary, or renal changes, is not infrequently caused by the presence of a goitre. The hypertrophy may be caused in one of two ways: (1) By mechanical interference as the result of extension of the enlarged thyroid through the upper aperture of the thorax. An intrathoracic goitre may cause pressure on the blood-vessels of the thorax or it may interfere with the respiratory act and thus throw additional work on the heart. Einthoven noted considerable pressure on the trachea in 15 per cent. of 100 cases. It is to be borne in mind, however, that goitre stenosis does not necessarily produce a goitre heart as one heart may resist the pathological effects while others do not.

(2) The most common form of cardiopathy resulting from goitre is that known as the thyrotoxic heart. This may be caused by any kind of a goitre but is seen in its most marked form in goitres associated

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[1] *Boston Med. and Surg. Jour.*, June 16, 1910.

[2] *Berl. Klin. Woch.*, September 25, 1912.

with thyroid intoxication, particularly in the exophthalmic type of the disease.

Morbid Anatomy.—The essential feature of goitre heart is enlargement. The hypertrophy may, for a time, be limited to the left ventricle, but later all of the chambers become hypertrophied and dilated. As already stated the hypertrophy and dilatation occur independently of any of the usual causes. In addition to the enlargement of the chambers, the endocardium may be thickened, either diffusely or in patches and this thickening may extend, in places, some distance into the underlying heart muscle. In common with the dilatation and evidences of failing compensation due to other causes, goitre hearts often contain mural thrombi and also give rise to hemorrhagic infarctions into the viscera, particularly the lungs and kidneys.

Symptoms.—When the heart enlarges as the result of mechanical interference the cardiac changes occur insidiously. The first noticeable symptom is, as a rule, shortness of breath and as time goes on this is increased by exertion. In some cases the first symptom is a change in the voice. Later the patient may complain of vertigo, headache, flushing of the face and annoying attacks of palpitation. With the onset of dilatation the symptoms and signs of broken compensation appear; the lungs become congested; the heart's action is constantly rapid and there may be, in addition, arrhythmia; finally edema makes its appearance.

In the thyrotoxic cases the first and the most constant cardiac manifestation is an increased heart action. Not only is the heart action increased in frequency but in intensity as well. The rapidity of the heart's action may come on gradually or it may occur in paroxysms. A very distinctive feature is the fact that the tachycardia is oftentimes caused by the slightest exertion, the pulse-rate quickly running up to 120, 150 or even higher.

Although tachycardia has long been recognized as one of the cardinal symptoms of Graves' disease it is only recently that the profession has come to realize that tachycardia arising as the result of exertion or emotional stress, is often a manifestation of unsuspected *hyperthyroidism*. During the past two or three years the examination of soldiers brought to light many of these cases of hyperthyroidism. The striking feature in nearly every instance is the tachycardia which occurs alike in recruits presenting themselves for initial examination and those who later have had their nervous and circulatory equilibrium upset. In these cases the heart rate is nearly always increased by exercise. Arrhythmia is rarely present. In addition to the tachycardia there is usually a marked throbbing of the superficial arteries and a capillary pulse may be simulated. The patient may complain also of alternate flushing and paling and may have attacks of faintness or dizziness.

In addition to the cardiac manifestations there may be marked nervous symptoms in the form of epileptoid attacks, outbursts of rage, crying, etc. These emotional outbursts are apt to be followed by considerable physical and mental prostration. Brooks states that in about one-third of the cases he encountered there was evidence of hypertrophy of the thyroid. In many others there was a history of goitre in the family, particularly on the maternal side. In others the family history revealed the presence of hysteria, insanity or perversions.

A condition presenting much the same picture as hyperthyroidism is that known as the *"effort syndrome."* The latter is characterized by tachycardia, breathlessness, precordial pain, faintness and exhaustion following exercise. No difficulty is encountered in distinguishing between the two conditions if the tachycardia and symptoms are associated with tumor, enlargement of the thyroid or one or more of the eye signs associated with Graves' disease. In many cases, however, it is almost impossible to determine which of the two conditions is responsible for the cardiac symptoms. Warfield[1] states that a past history of precordial pain, exhaustion, and faintness is almost positive proof that the "effort syndrome" is present. *Latent tuberculosis* may produce much the same phenomena. It is to be distinguished from hyperthyroidism and the "effort syndrome" by reason of the rise in temperature which follows the exercise, no such elevation occurring in the other conditions. The adrenalin test is also of service in doubtful cases. The injection of 7½ minims of adrenalin (1–1000) brings to light the evidence of hyperthyroidism or accentuates doubtful symptoms, if this condition is the underlying cause of the trouble.

In patients in whom the thyroid gland is enlarged a rapid pulse should always arouse the suspicion that the tachycardia is thyrotoxic rather than cardiac in origin. In additon to the tachycardia, of which the patient may or may not be conscious, there is often a complaint of palpitation on exertion, precordial distress and throbbing of the vessels in the neck. The evolution of the hypertrophy of the heart differs in no way from that due to well-recognized causes. Sooner or later dilatation occurs and the patient then presents the usual phenomena of failing compensation. Many of the cases when first seen complain of shortness of breath, palpitation, precordial distress and edema of the lower extremities.

Physical Signs.—*Infection.*—The apex beat of the heart is usually displaced to the left and the cardiac impulse is forcible and heaving. The superficial vessels, especially in the neck, pulsate strongly and a capillary pulse may be present. The cardiac impulse may be so forcible as to shake the head or even the whole body of the patient. A pulse tracing readily distinguishes the condition from aortic regurgitation.

Palpation.—The cardiac action is felt to be both rapid and forcible. In the early stages the pulse is regular, readily compressible and often dicrotic. In the later stages when myocardial changes cause dilatation Krumbhaar[2] found that the electrocardiograph showed various forms of arrhythmia. Blackford and Willus[3] in a study of *auricular flutter* found that condition most frequently associated with exophthalmic goitre. If the arrhythmia is caused by thyroid intoxication it may disappear with the removal of the intoxication. Palpation of the thyroid gland may show expansile pulsation.

Percussion.—The first change is noted on percussion in an extension of dulness to the left due to hypertrophy of the left ventricle. In the later stages when the right ventricle becomes dilated the area of cardiac dulness will extend from 1 to 4 centimeters beyond the right pleural border. If the heart outline is determined before and after exercise it will often be noted that even slight exertion increases the area of dulnes very considerably. Crotti[4] considers that a heart that shows an increa in the area of dulness after exertion indicates the presence of myocarditis. Operation should be attempted with great care in such cases.

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[1] *Trans. Assoc. Amer. Physicians*, 1919.

[2] *Amer. Jour. Med. Sc.*, Feb., 1918.

[3] *Archives of Internal Medicine*, Jan., 1918.

[4] *Ohio State Med. Jour.*, Feb. 15, 1912.

Auscultation.—In the early stages nothing abnormal may be noted. Very frequently, however, an apical systolic murmur is present. This is a common finding in cases of hyperthyroidism after the exercise test. In the later stages of thyrotoxic heart the auscultatory findings are those of chronic myocarditis. The sounds may be sharp and accentuated, lacking muscular tone, or feeble and irregular. A systolic murmur, due to relative insufficiency, is often present.

A systolic murmur may be heard also in the vessels of the neck and over the thyroid gland. A continuous hum is often to be heard above the clavicles.

Diagnosis.—In patients having a goitre, with or without the Graves syndrome, the pressure of cardiac disturbance points to a thyrotoxic origin. And even when a goitre is not demonstrable hyperthyroidism is to be suspected if marked cardio-vascular disturbances are produced by exercise and these persist after rest.

THE HEART AND UTERINE MYOMATA

Much has been written concerning a causal relationship between myoma of the uterus, and cardiac lesions. The association of functional or even organic heart disease, and the unexplained occurrence of sudden death before or after operations for "fibroids" has led to much speculation on the subject. Brown atrophy, fatty degeneration, cloudy swelling of the myocardium have been described together with arrhythmia and valvular disease.

The subject was carefully studied and reviewed by McGlinn[1] who came to the conclusion that "a definite entity of fibroid heart cannot be sustained." The cardiac sclerosis occurring in myoma cases during middle, and advanced life are simply a part of a general arteriosclerotic process. Cardiac hypertrophy and dilatation may result from pressure upon the pelvic circulation. Myocardial changes may result from anemia, due to hemorrhages, from infections or perhaps from secondary degenerations of the tumor. The association of lesions between myomata and the heart are simply coincidences. As a specific entity the "fibroid heart" does not exist.

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[1] *Surgery, Gynecology and Obstetrics*, 1914, Feb., 180.

CHAPTER XXVII

ENDOCARDITIS

ACUTE ENDOCARDITIS

While any portion of the endocardium may become the seat of i matory changes the term "endocarditis" is usually understood to r the valves. It is to be borne in mind, however, that the inflamn process may involve the mural endocardium independently or as the of extension from the valves. In like manner an acute aortitis ma as the result of a concomitant infection of the semilunar valves.

Two types of acute endocarditis are commonly considered, n simple and malignant. It is generally recognized, however, th classification is an arbitrary one and that no sharp dividing line drawn. Simple acute endocarditis has been, and is still sometin ferred to as benign. The designation is a bad one and should employed, as it conveys the impression that a large group of cases of carditis may suffer from no serious after-effects. It cannot be too st impressed upon the mind that simple endocarditis is always fraugh danger and that more people ultimately die as the result of su occurrence than is the case with the more serious malignant or in type of the disease. Although we clearly recognize the fact that t tinction between the simple and the malignant types of the dis largely one of severity, there are, at the same time, certain etio and clinical differences which makes a separate description of th processes advisable.

ACUTE SIMPLE ENDOCARDITIS

Etiology.—This type of the disease occurs more often in chil and adolescence than in any other period of life, largely because frequency of attacks of *acute rheumatic fever* in early life. So com does acute endocarditis develop as the result of rheumatic fever t is sometimes termed rheumatic endocarditis. Prior to the age of pu rheumatic attacks are almost constantly associated with the en dial lesions. The following table compiled by Cowan[1] indicat extraordinary frequency with which the two conditions co-exist:

	Cases of acute rheumatism	Cases of endocar
Gibson	325	161
Macrae	300	105
Church	889	494
Moore	100	90
Latham	136	74
	1750	933

These figures, which show that 53.3 per cent. of the cases develo acute endocarditis, serve to emphasize the important relationshi acute rheumatic fever bears to cardiac disease. As a rule, the endocarditis develops during the first attack of rheumatism and the valvular damage is apt to be increased if subsequent attacks occur. The endocarditis may manifest itself as early as the second day of the rheumatic attack or it may be delayed until the inflammatory symptoms have entirely subsided. The frequency of the endocarditis bears no relation to the severity of the arthritis. An extremely severe attack of rheumatism may not involve the endocardium; on the other hand, a trifling and mild attack may cause serious endocardial mischief.

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[1] "Diseases of the Heart," 1914.

Closely allied to the rheumatic type of endocarditis is that which follows attacks of *tonsillitis*. The importance of tonsillitis as a cause of endocarditis is now fully recognized. Very often the tonsillar attacks are very mild and pass for nothing more than a slight sore throat. It is quite possible that many cases of endocarditis, in which there is no apparent etiological factor, have had their origin in attacks of mild and unrecognized tonsillitis or acute rheumatic fever. In children suffering from a chronic valvular lesion, careful questioning will sometimes reveal a history of vague and fugitive joint pains which at the time of their occurrence caused little or no concern.

Next to rheumatic fever the most frequent cause of endocarditis is *chorea*. "Valvular lesions are extremely common in choreic patients, and were present in 262 out of 829 patients who were examined during or after illness" (Cowan).

Any of the acute infectious diseases may give rise to an endocarditis, but with the exception of croupous pneumonia such an occurrence is not common. Not infrequently acute endocarditis is noted in phthisis, cancer, gout, diabetes and Bright's disease. The condition is practically always a secondary one and even in those instances in which a primary infecting focus cannot be discovered it is now held that this undoubtedly is present. Recent bacteriological studies indicate that in the majority of cases of active endocarditis a streptococcus bacteremia is present.

Morbid Anatomy.—Except when the endocarditis arises during intra-uterine life the valves of the left heart are the ones which are almost invariably affected. When a valvulitis arises as the result of some infectious or toxic agent, the inflammatory process occurs in certain definite areas. The mitral leaflets are more frequently involved than the aortic. Sibson has offered the explanation that the increased liability of the mitral valve is due to the fact that the edges of the leaflets of this valve are closed with much greater tension than is the case with the aortic valve. The mitral leaflets are subject also to a wider range of play—wider excursions, therefore, produce more wear and tear. The endocardial changes as they occur in the valves take place on the auricular surface of the mitral and the ventricular surface of the aortic due to the fact that these surfaces are bathed by the blood stream. The lesions are located just behind the free edges of the valves.

The first evidence of the inflammatory change is a slightly swollen appearance of the affected area and a faint hyperemia due to the increased vascularity of the part. This is followed by a shedding of the endothelial and subendothelial cells, which gives rise to a grayish, opaque appearance. Over the site of these degenerated areas fibrin is deposited in the form of small bead-like excrescences or granulations (endocarditis verrucosa). The size of the growths vary greatly; they may become sufficiently large to prevent closure of the valve or obstruct the blood stream. Tangled in the meshes of the fibrinous deposits bacteria are found, although this is more commonly the case in the malignant than the simple form of the disease. In the majority of instances of simple endocarditis no bacteria are found; this is especially true of the affection as encountered in the chronic wasting diseases.

In addition to the involvement of the valves the chordæ tendineæ may be implicated and later become shortened as the result of contracting scar tissue.

It is now recognized that an acute endocarditis is almost always accompanied by myocardial change. The myocardial change may be general or it may be limited to, or most marked in, the muscle tissue at the base of the affected valve. The myocardial change may be very mild and trivial and give rise to no evidence of its existence. On the other hand, disturbances of rhythm may occur as the result of implication of Tawara's node or the bundle of His. In a very fair proportion of cases of acute endocarditis there is also an associated pericarditis.

Simple acute endocarditis is of itself not serious. The acute inflammatory stage is rarely intense enough to destroy tissue and emboli rarely arise from the vegetations. Its great danger lies in the after-results. With the subsidence of the acute stage granulation tissue is formed and this organizes into scar tissue. With the retraction of the scar tissue the edge of the valve may be curled back giving rise to incompetency or the leaflets may become ankylosed producing a stenosis. Another very serious danger is the fact that these fibrous patches on the valves are very prone to become reinfected with either the simple or malignant form of the disease. Malignant endocarditis is very commonly engrafted on an old chronic valvular defect.

Symptoms.—There are no distinctive symptoms of acute simple endocarditis; indeed, they may be wanting entirely. In a very large proportion of cases the cardiac lesion is masked by the primary infection. Fever is probably the most important symptom referable to the endocarditis. It is not always easy, however, to determine its significance as fever is already present as a result of the rheumatic attack, pneumonia or other acute infection. One should be suspicious of endocardial involvement if the temperature changes in character or if, after a period of normal temperature, fever again develops. In some cases there may be a sense of precordial oppression or actual pain and in others there may [illegible] palpitation of the heart.

Physical Signs.—I have already alluded to the great frequency with which acute endocarditis is associated with rheumatic fever. As an etiological factor it overshadows all others, especially in young children. It is essential to keep this in mind and to take the precaution of examining the heart daily as in the absence of symptoms a cardiac lesion may very easily escape notice. Owing to the very serious after-effects which may arise, even from a trivial lesion, it is incumbent upon us to detect the trouble as early as possible so as to prevent any undue strain being put upon the heart until the acute stage of the disease has passed.

Inspection.—In the presence of acute rheumatic fever inspection of the precordium should be carefully made each day as by this one procedure most valuable information may be obtained. While no abnormality may be noted throughout the course of the disease there will often be seen a gradual displacement of the apex beat beyond the nipple line; or the cardiac impulse may be forcible; or instead of a forcible heaving impulse it may be diffuse and wavy in character.

Palpation.—This will confirm the character of the impulse noted on inspection. In addition, the shock of both the first and second sounds may be felt.

The character of the *pulse* should be carefully noted as, in not a few cases, it furnishes the only clue pointing to a cardiac lesion. The pulse may be unduly accelerated in proportion to the degree of fever or it may remain high after the primary infection has subsided. Cowan has pointed out that the administration of the salicylates will often slow the pulse rate and that after these drugs are discontinued the pulse rate will increase in frequency if an endocarditis is present. In some cases the pulse is unduly slow. Irregularity of the pulse is also of great importance. The irregularity may be due to a temporary heart block, to extrasystoles or auricular fibrillation.

Percussion.—The information obtained by percussion of the precordium, in cases of acute endocarditis, is not great. There may be some increase in the area of cardiac dulness upward and to the left and occasionally to the right.

Auscultation.—The most constant sign and, at the same time, the most reliable evidence of acute endocarditis is a systolic murmur at the apex. The greater frequency of apical as compared to aortic murmurs is due to the fact that in the great majority of cases of simple endocarditis the rheumatic infection affects by preference the mitral valve. The murmur may occur within a few days of the onset of the rheumatic attack or it may not be heard until convalescence is established. Then again, it may be heard first some weeks or months after complete recovery. In other instances a murmur may be heard during the height of the febrile attack, then disappear, and again recur as the result of a chronic endocarditis. In such cases it is possible that the vegetations interfere with proper closure of the valve and as these are absorbed the murmur disappears, or, what is more probable, the murmur is caused by the associated myocarditis. The recurrence of a permanent murmur is caused by contraction of the scar tissue which distorts the leaflets or causes their adherence, with the result that a leakage or obstruction is caused.

The time of the murmur in acute endocarditis is always systolic; it is only when a chronic endocarditis is established that a murmur indicative of obstruction is heard. While the first indication of trouble may be a soft blowing murmur it will be noted, if the heart is examined from day to day, that the initial change is usually a blurring of the first sound; later the first sound becomes roughened in character or sharply accentuated and finally a murmur is heard. Associated with the murmur is an accentuation of the pulmonic second sound. If the murmur is truly organic, it will be heard best at the apex and is transmitted towards the left axilla. In determining whether the murmur is organic or functional in character it must be borne in mind that in acute rheumatic fever a marked secondary anemia is sometimes rapidly established and this may cause a functional systolic murmur at the apex or base of the heart. Very often it is not possible at once to decide the question and it is only later when other signs appear that one can be certain that the murmur is organic in nature. While an organic murmur may vary in intensity from day to day, one of its characteristic features is to gradually become more and more evident.

In the presence of fever it is not uncommon to hear a systolic murmur at the pulmonic area which is without significance. Finally there may be a cardio-respiratory murmur (see p. 254).

Less commonly the changes described above are heard over the aortic area or both valves may be involved simultaneously. In some instances there may be a pericardial friction rub.

ACUTE INFECTIOUS ENDOCARDITIS

(Septic, Malignant, Ulcerative or Pernicious Endocarditis)

Etiology.—This form of the disease rarely develops in the course of acute rheumatic fever or chorea. In the great majority of instances it arises as the result of a septic wound, puerperal sepsis, erysipelas, a gonorrheal infection or some inflammatory disease of the lungs, notably pneumonia. Some one of the various types of the streptococcus is by far the commonest organism recovered from the lesions. Among the rarer bacteria may be mentioned the bacillus of diphtheria and influenza, the meningococcus and staphylococcus.

Malignant endocarditis is rare in young children; the great majority of cases occur in young adults or those in middle life. This is probably accounted for by the fact that the septic form of the disease very frequently occurs in valves previously damaged by an attack of acute rheumatic fever.

Acute infectious endocarditis is most commonly due to hemolytic streptococci, pneumococci, staphylococci, the influenza bacillus and the gonococcus. Occasionally other organisms are the exciting causes.

Morbid Anatomy.—The initial changes in the infective type of endocarditis may present the same appearance as is seen in the simple form of the disease. Usually, however, the lesions become more marked; the excrescences are much larger and in extreme cases may be the size of a cherry (vegetative endocarditis) (see Figs. 393 and 394). The largest vegetations are apt to occur when the infecting organism is either the pneumococcus or the gonococcus. If the vegetations are broken off, an ulcerated surface is left which may perforate the valve or produce a valvular aneurism.

In another type of the disease, known as *endocarditis ulcerosa*, the first stage shows a small, somewhat opaque, yellow patch, with a slightly uneven surface. In parts of such patches there may be a loss of substance. Over these ulcers there may be deposited reddish-gray thrombotic masses, which, when removed, will show the underlying tissue to be swollen, ulcerated and yellowish in color (Adami and Nicholls).

In some instances the infection may spread to the wall of the heart where similar changes occur in the mural endocardium (see Fig. 395).

Cultures taken from the ulcers or vegetations will show large numbers of microörganisms which are often extremely virulent. A histological examination of the affected area will also show countless bacteria.

According to Horder[1] a culture of a pathogenic organism can be obtained in 90 per cent. of cases of infective endocarditis. In addition i a very high percentage of cases, the infecting organism can be obtain from the circulating blood. In by far the largest proportion of cases t cause of the trouble is a streptococcus.

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[1] *Quarterly Jour. Med.*, vol. ii, p. 289.

The frequency with which the different valves are affected is as folrs: the mitral, aortic, mitral and aortic together, the tricuspid and the lmonary. Involvement of the mural endocardium is also frequently ed.

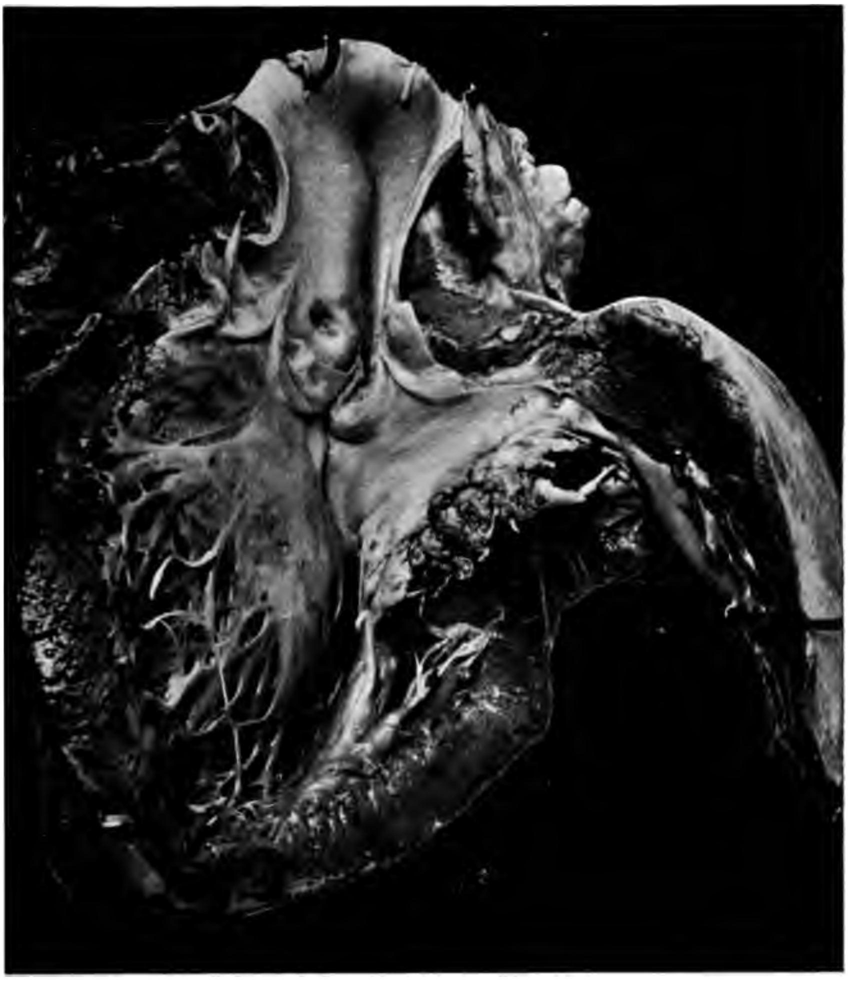

Fig. 393.—Acute infective mitral and mural endocarditis. The mitral valve is covered ith large vegetations. Several of the chordæ tendineæ have been destroyed by ulceraon, only short unattached stumps remaining. Vegetations are also seen on the mural ndocardium. *(Specimen from the Pennsylvania Hospital.)*

One of the most striking features of malignant endocarditis is the ccurrence of an *embolism*. This is not surprising when we recall the oft, friable nature of the valvular vegetations. As the lesion progresses nd necrosis sets in small pieces are whipped off by the blood current nd carried to some distant point. The viscera most commonly involved re the spleen, kidneys and brain, but no portion of the body is exempt.

Occasionally mycotic aneurisms involving the larger vessels ha
origin in this way. If the valves in the right heart are involv

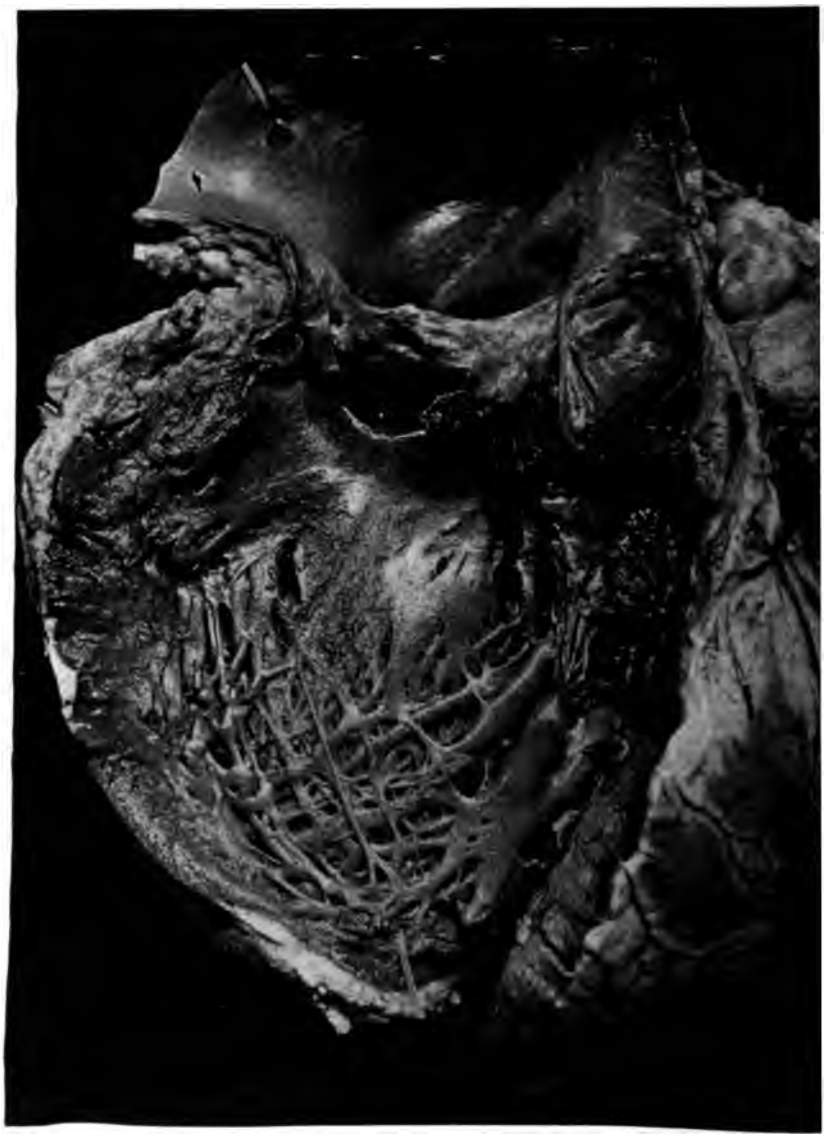

FIG. 394.—Acute infective endocarditis. Showing extensive ulceration of
valves. About the center of the aortic orifice a match-stick projecting from a th
mass indicates the course of the perforation in the aortic leaflet. (*Specimen from*
delphia Hospital.)

may be infarcts in the lungs. Owing to the infective nature of the
the resulting infarcts commonly terminate in abscess formation.

In some instances the embolic phenomena are absent.

Symptoms.—The clinical features of septic endocarditis do not lend themselves readily to description. In some instances the true nature of the trouble can be surmised without much difficulty, but in a very considerable proportion of cases the endocardial lesion is masked by the primary disease, such as puerperal sepsis, pneumonia, etc.

"It may be questioned whether it is worth while to consider the protean aspects of this infection under diseases of the heart, since the manifestations are those of septicopyemia; and in a great majority of all cases the features of the general infection dominate the picture" (Osler).

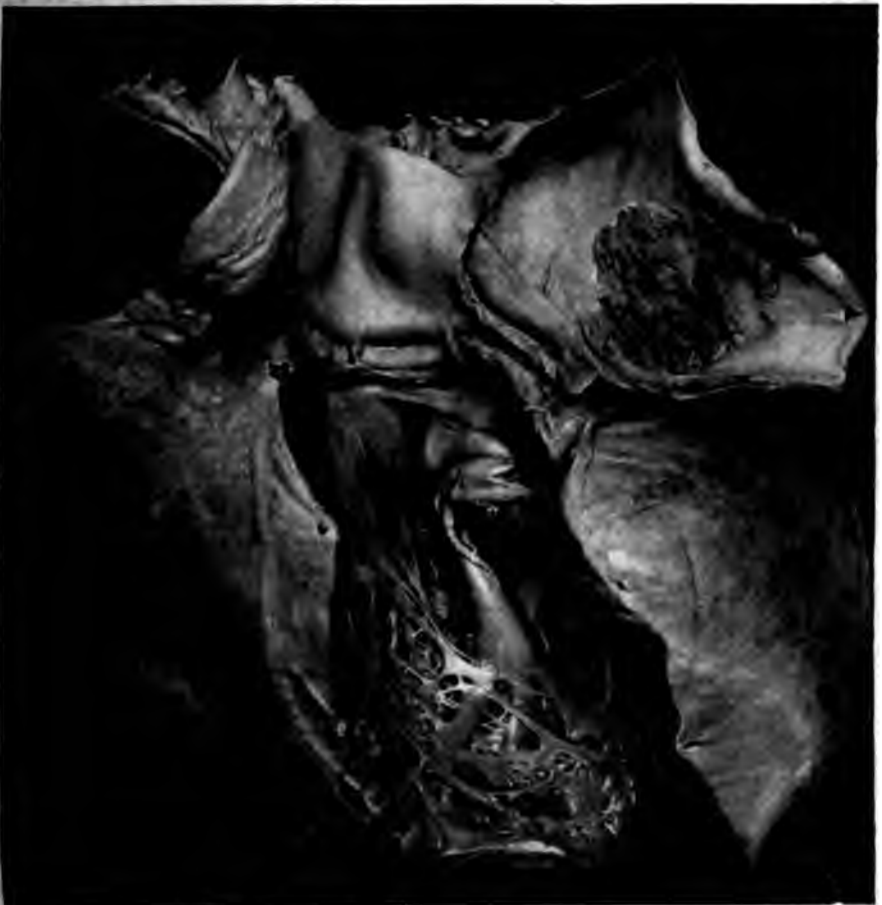

FIG. 395.—Acute mural endocarditis.

Although the onset is varied and often ill-defined, the process when once established presents, in the majority of cases, certain general features. These are, an irregular type of fever, sweating, delirium, progressive anemia, loss of weight, a change in the character of the cardiac murmurs and the occurrence of emboli.

The character of the fever is variable. It may be remittent, intermittent or continued in type (Fig. 396). In some cases it is paroxysmal, recurring every two or three days with chills and drenching sweats thus resembling malarial fever. The fever may be moderate or very high but no matter what type it assumes is apt to be irregular. A moderate amount of fever of the continued type is commonly associated with endocarditis superimposed on a chronic valvular lesion. Rarely a case of septic endocarditis is encountered which is afebrile.

The *embolic phenomena*, although sometimes absent, are the most certain indication of septic endocarditis. The spleen is the organ most frequently involved and the emboli may be confined to this viscus alone.

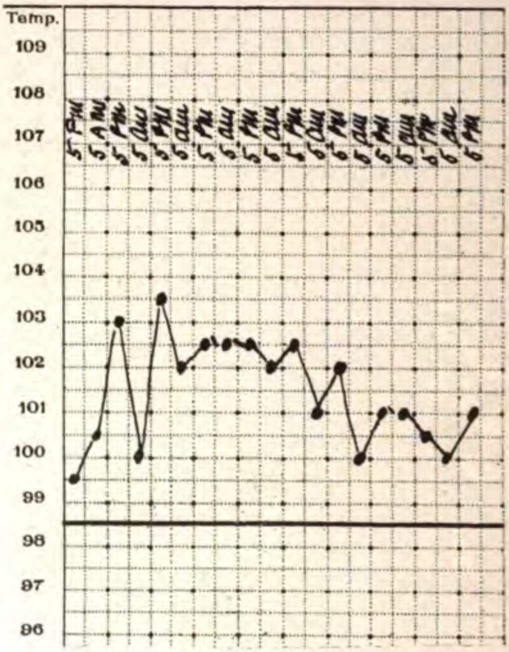

FIG. 396.—Malignant endocarditis.

An embolus in the spleen manifests itself by sudden pain in the left side, tenderness on pressure and enlargement of the organ. When the kidneys are affected there are renal pain and bloody urine. Convulsions, hemiplegia, or aphasia may follow an embolus in the brain and visual defects may occur if one of the retinal vessels is involved. Small petechial spots may appear in the skin as the result of minute emboli. Occasionally the embolus blocks one of the larger vessels in an extremity and gangrene ensues. If the lesion is right-sided, emboli may be carried to the lungs.

In all cases of suspected septic endocarditis, providing the facilities are available, a blood culture should be obtained. A lumbar puncture should also be made. A leukocytosis is usually present.

Generally speaking, septic endocarditis presents itself in one of three ways: (1) The phenomena may be almost entirely cardiac in character:

) a pyemic form in which there is a demonstrable focus of infection; and
) a typhoid form in which the infecting focus is not apparent.

1. The *cardiac form* is the one which is most easily recognized. In is type of the affection, the patient, already suffering from a chronic ılvulitis, develops fever of a continued or slightly irregular type, an crease in the cardiac symptoms, and in addition there may be an em-ılism. The attack may be very acute or may be relatively mild and rsist for many weeks. Recurrences are common and recovery often curs.

2. In the *pyemic form* the endocarditis is but an incident in the general fection. Its recognition is always attended with considerable difficulty ıd it often escapes notice entirely owing to the absence of a murmur or her cardiac signs. Chills, sweating, irregular fever, anemia and wasting e characteristic of a general sepsis and while involvement of the endo-ırdium may aggravate these symptoms, one cannot make a diagnosis of ıdocarditis in the absence of a murmur, cardiac dilatation or embolic ıenomena. If, in the course of general sepsis following an external ound or the puerperium, there develops a loud murmur or a murmur eviously present increases in intensity or changes in quality, the assump-on that the heart valves have been included in the infection is nearly ways warranted. Dilatation of the heart in the course of septicemia so points to cardiac involvement. In the absence of cardiac signs the currence of an embolism furnishes ample evidence as to the existence an endocarditis.

3. The *typhoid form* is by far the most common type of malignant en-ocarditis and the one attended with the greatest difficulty in its recogni-on. In this form we lack, as a rule, the presence of an old valvulitis r a demonstrable focus of infection to arouse our suspicions. The llowing symptoms are common to both typhoid fever and septic endo-ırditis: a continuous type of fever, progressive exhaustion, anemia, a low ıuttering type of delirium, enlargement of the spleen, diarrhea, a dry ngue and sordes. In addition, petechial and other skin rashes are not ıfrequent. While a patient with septic endocarditis may not have all hese symptoms, the occurrence of several of them may point so strongly o typhoid fever that the true nature of the trouble is not thought of. In ther cases cerebral symptoms may predominate and lead to a diagnosis f cerebro-spinal or basilar meningitis. Cardiac signs and symptoms may e wanting entirely or they may be very indefinite in character. Embolic henomena are less common in this type than in the others; their occur-ence often clears up an obscure case at once. The disease lasts for from wo or three weeks to three months.

This type of endocarditis is very frequently due to a pneumococcus, or nococcus infection.

Physical Signs.—Physical signs referable to the heart may be absent very indefinite. The patient is lethargic, the face is pale and sunken ıd the general appearance is indicative of some severe infection. Unless ere has been present a chronic valvulitis the physical signs are almost tirely auscultatory although in the terminal stages evidences of cardiac atation may be made out. Mackenzie lays great emphasis on the pulse doubtful cases associated with pyemia and puerperal septicemia. In ese cases the heart may show but little change except that the heart ınds are feeble. The pulse is small, soft, easily compressed and not necessarily rapid, although the rate is apt to be higher than the degree of fever warrants.

The presence of a murmur is the determining factor in most cases. The murmur may be very faint and repeated examinations may be necessary to determine its presence. One of the characteristics of a murmur occurring in malignant endocarditis is its variable character. It is apt to change both in quality and intensity from day to day. The sudden appearance of a musical murmur is strongly indicative of septic endocarditis. A murmur of this character is caused by a large and projecting vegetation. If in the course of an obscure infection a murmur due to chronic valvulitis undergoes changes or, in addition to the mitral murmur, a diastolic murmur at the aortic area develops during the course of the disease, one should be alert as to the possibility of an infectious endocarditis.

Diagnosis.—In that group in which the symptoms are largely cardiac in character the diagnosis is fairly easy, especially if there is known to be a chronic valvulitis already present. In such cases the occurrence of fever, the presence of a murmur which is subject to changes from day to day and the appearance of embolic phenomena point unmistakably to septic endocarditis.

In the pyemic form of endocarditis the diagnosis will depend largely on the detection of valvular mischief. The endocarditis is simply an incident in the general septic process and unless a murmur develops or embolic phenomena occur the cardiac element will pass unnoticed. As previously stated a small, soft and easily compressed pulse is significant in these cases of general sepsis.

When the septicemia is characterized by paroxysmal attacks of chills, sweating and fever the condition may be mistaken for *malaria*. An examination of the blood and the presence or absence of the malaria plasmodium or a leukocytosis would readily decide the question. The presence of a positive blood culture is of no value in determining the presence of endocardial involvement, although such a finding demands that the heart be carefully examined.

The typhoid form gives rise to the greatest confusion and in any given case it is often most difficult to distinguish between malignant endocarditis, *typhoid fever* and *acute miliary tuberculosis*. Marked prostration, anemia, wasting and the so-called typhoid state may occur in all three. The temperature seen in true typhoid may be atypical; on the other hand, that in endocarditis and miliary tuberculosis, while subject to many variations, may closely resemble that seen in typhoid. Even with the presence of a mitral murmur one cannot be certain that it is not functional in character, owing to the exhaustion and anemia common to all these conditions. In the absence of definite cardiac signs the diagnosis may be cleared up by a blood culture or lumbar puncture. If malignant endocarditis is present, a blood culture will show, in a fairly large percentage of cases, the infecting organism, usually a streptococcus, pneumococcus or gonococcus. In typhoid fever, blood cultures if taken early in the disease will frequently show Eberth's bacillus. A positive Widal reaction speaks for typhoid but not infrequently it also occurs in miliary tuberculosis. The latter condition is notoriously difficult to recognize in adults although relatively easy in children. The lungs may be studded with minute tubercles and yet produce no physical signs until the terminal stages.

horoidal tubercles are frequently cited as an aid in the diagnosis but I ave never seen a case in which they were recognized. In malignant ndocarditis there is usually a leukocytosis, while in typhoid fever and iliary tuberculosis there is a leukopenia or a normal white count. lthough an attack of the typhoid form of endocarditis is often prolonged r several months the diagnosis, in many cases, may never be cleared up tisfactorily; in others, the appearance of embolic phenomena renders the ture of an obscure infection clear (see also p. 385).

Subacute Infectious Endocarditis

This condition, first described by Osler, is also known as subacute cterial endocarditis, subacute streptococcus endocarditis, chronic ma- gnant endocarditis, chronic ulcerative endocarditis and endocarditis nta. In this form of the disease the patients are the subjects of a ronic valvulitis which is often well compensated and causes little or trouble. A fresh infection is superimposed on this old lesion, thus rming the focus of a subacute or chronic septicemia.

The condition is due to anhemolytic streptococci in over 95 per cent. f the cases. The majority of the remaining cases is due to the influenza acillus. Other organisms, particularly the gonococcus, may rarely ause a subacute clinical picture (Libman). Blood cultures made during he bacterial stage almost always show the presence of anhemolytic strep- ococci or influenza bacilli.

The duration of the disease is from a few months to a year and a half r two years. Occasionally recovery takes place, but in the great major- ty of cases a fatal issue ensues.

Morbid Anatomy.—Libman, who has had a large experience, describes he pathological changes as follows: The mitral valve is the seat of the esion in about two-thirds of the cases and the aortic in one-third. In- olvement of the right side of the heart is rare. A striking feature is the nvolvement of the wall of the auricle and the chordæ tendinæ. The egetations are yellowish, greenish, pinkish or reddish in color and vary reatly in size. When the aortic valve is involved, the vegetations re usually few and of small size, although occasionally there may be an normous green mass which almost blocks the orifice (see Fig. 397). As hey grow older the vegetations become firmer and grayish in color.

When the mitral valve is involved, the vegetations spread up on left osterior wall of the auricle (see Fig. 397). The chordæ tendineæ also ay be covered with vegetations and may become torn so that the ends ay lie loose near the papillary muscles. When the aortic valve is the at of the disease the vegetations have a tendency to extend down over e endocardium and over the ventricular aspect of the aortic flap of the itral valve. Occasionally an aneurism of the valve leaflets may develop.

Cerebral embolism, uremia, pneumonia and progressive anemia are e common terminations.

Symptoms.—The onset is often insidious and without definite symp- ms. Very often, however, the initial symptoms are definitely referred the heart, as for instance, shortness of breath on exertion, palpitation, d cough which is sometimes attended with blood-streaked sputum.

Fever is present in all cases (Fig. 398). It is often slight but in the jority of cases is high and intermittent in the late stages of the disease.

If splenic infarcts occur, the fever is apt to be high and intermittent. Chills and sweating may occur in association with the fever. Loss o weight and extreme weakness take place in most cases.

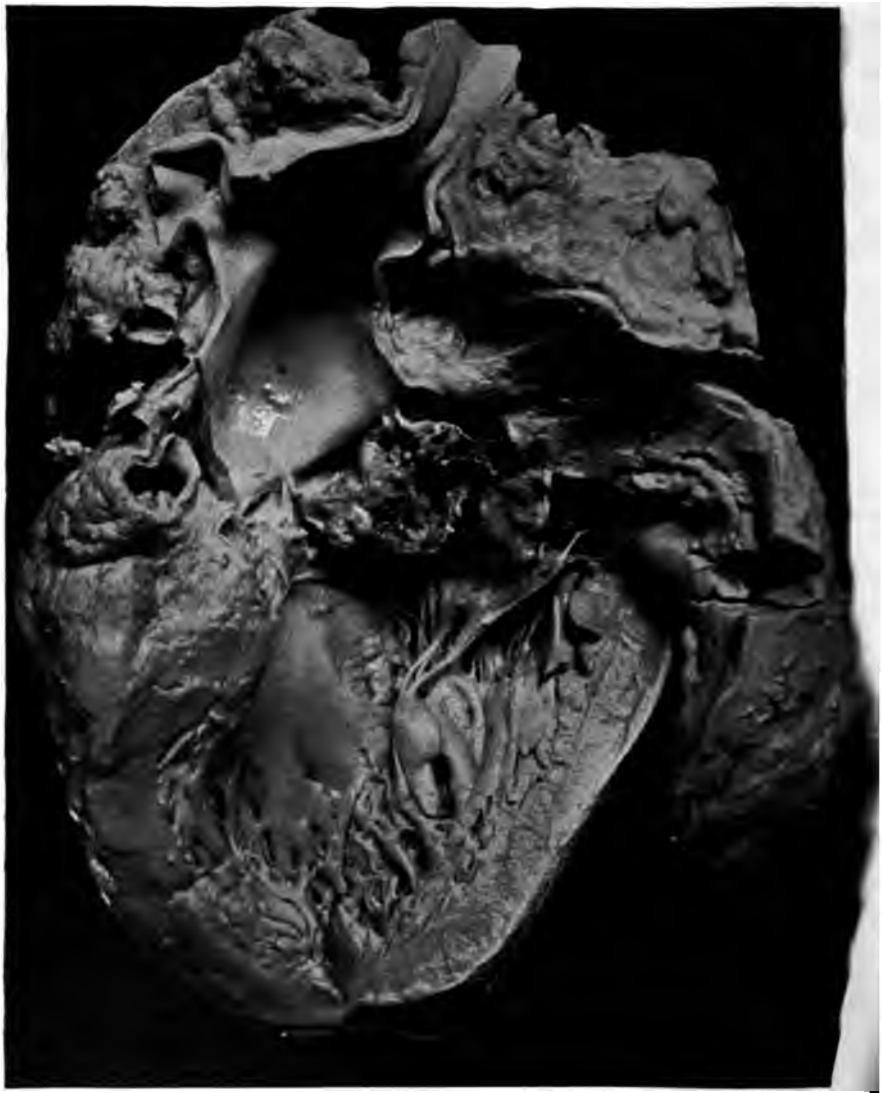

Fig. 397.—Subacute infective-endocarditis. Showing large thrombotic masses almost completely occluding the aortic orifice. There is marked left ventricular hypertrophy, and sclerotic patches are seen on the aorta above the sinus of Valsalva.

Precordial pain or a sense of oppression in the chest is often noted. Pain in the joints is not uncommon and may be associated with slight transitory swellings. A purpuric eruption may be present also. Libman has called attention to an important diagnostic sign, namely, tenderness over the lower part of the sternum. The lightest tap with the percussing finger may cause marked pain.

An important diagnostic feature is the presence of small, painful erythematous nodules in the skin of the hands and feet. These cutaneous nodes rarely occur except in this form of endocarditis and are considered by Osler as being pathognomonic. Petechiæ in the skin and conjunctival mucous membrane are frequently noted.

Hematuria occurs in a few cases and albumen and casts are found from time to time. The leukocytes are not often increased in number. A characteristic feature is the presence of a more or less marked *anemia* which, in many cases, is progressive. Libman has emphasized the peculiar sallow or waxy color of the face in these cases. Later the face may assume a coffee-colored tint. In cases which become bacteria-free, the face and other portions of the body often assume a diffuse brown color. The spleen is enlarged in nearly all cases and is easily felt on palpation. In some instances the organ is enormously enlarged.

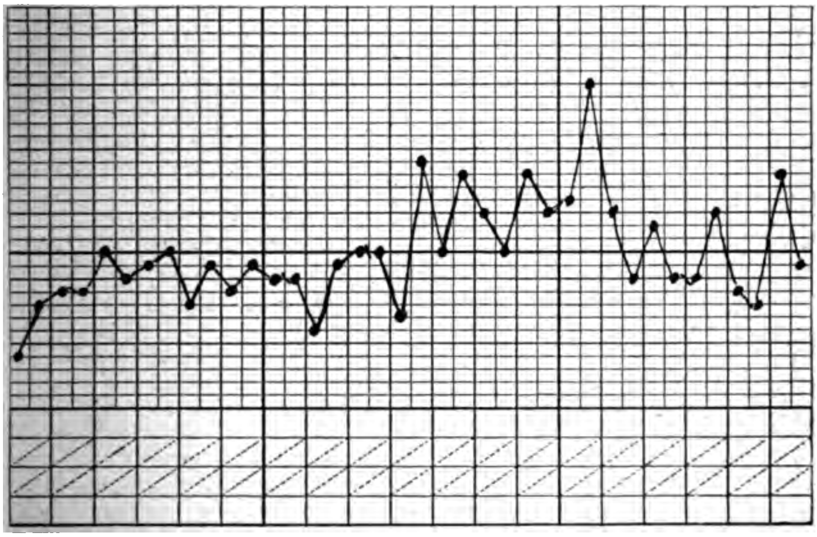

FIG. 398.—Temperature chart from a case of subacute infectious endocarditis.

Physical Signs.—Examination of the heart shows in every instance the presence of an organic murmur either at the aortic or mitral orifice, usually the latter. Aside from the murmur the heart may show no change throughout the course of the disease. This is especially true when the mitral valve is involved. If the aortic valve is primarily implicated, the cardiac changes are apt to be more noticeable. In some cases there are progressive cardiac changes, the heart gradually dilates, the character of the mitral murmur changes or there develops under observation a diastolic aortic murmur.

In addition to the bacterial stage Libman describes a bacteria-free healing and a bacteria-free healed stage. The latter is often characterized by tenderness over the sternum and a brownish discoloration of the face and other portions of the body. In some, slight symptoms occur intermittently; in others, nephritis develops and the patient dies of uremia; while in still others, the anemia persists and signs of failing compensation develop.

Diagnosis.—Subacute infectious endocarditis is to be thought of if a patient, the subject of chronic valvulitis, develops a prolonged fever, becomes anemic, gradually fails in strength and if during the course of the disease, develops small, painful erythematous nodules in the skin of the hands or feet. Osler emphasizes the fact that these changes may occur without any additional alteration in the heart. Individuals suffering from vague symptoms of the neurasthenic type are not infrequently the victims of a subacute bacterial endocarditis. Blood cultures should be made in all suspected cases.

In the bacteria-free cases the marked tenderness over the lower portion of the sternum and the diffuse brownish discoloration of the face and other portions of the body are important diagnostic points.

CHRONIC VALVULAR DISEASE OF THE HEART

Etiology.—Of the various manifestations of cardiac disease chronic valvulitis comprises, by far, the largest group. Statistical studies indicate that valvular disease represents about 80 per cent. of the lesions which may involve the heart. In considering valvular lesions it must be borne in mind constantly that it is the cardiac muscle that determines the functional capacity of the heart.

Chronic valvulitis, or, as it is more commonly termed, chronic heart disease, is slightly more frequent among females than males. This is to be ascribed to the greater frequency of *acute rheumatic fever* and *chorea* among females. Oille, Graham and Detweiler[1] emphasize the importance of *tonsillitis* as an etiological factor, especially in children and young adult females. Prior to the fifth year chronic endocarditis is uncommon but beyond this age period the incidence gradually increases. The majority of instances of cardiac failure resulting from a chronic endocarditis are encountered between the eighteenth and fortieth years. After middle life the incidence of cardiac failure due to valvular defects gradually diminishes. The Negro is apparently more susceptible to organic heart disease than the white man. Of 292 deaths from organic heart disease in the Charity Hospital, of New Orleans,[2] 181 were negroes and 111 whites.

The deformity of the valves is brought about in several ways: (1) Not less than half of the cases are the direct result of an acute endocarditis. In discussing the etiology of acute endocarditis it was shown that acute rheumatic fever was by far the most important factor leading to acute inflammatory changes in the valves and that a very large proportion of these acute cases terminated in chronic valvular defects. Closely allied to the rheumatic cases are those which follow chorea and tonsillitis. Of the other acute infectious diseases, any one of them may cause acute endocardial changes which terminate in a chronic lesion, although the number of cases having their origin in this way is not great. In a not inconsiderable number of cases, particularly when the mitral valve is involved, no etiological factor can be determined.

2. In quite a large number of cases of chronic valvular disease, es-

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[1] *Jour. Am. Med. Assoc.*, October 2, 1915.

[2] *Report of Charity Hospital*, 1917.

pecially in those past the middle period of life, we are unable to obtain a history of acute endocarditis. In these cases we have to deal with a form of endocarditis which is chronic from the beginning. Primary chronic endocarditis is caused by the same conditions that lead to general arterio-sclerosis. Inasmuch as the intimal lining of the arteries and the tissue covering the valves is exactly similar, every etiological factor favorable to the production of general arterio-sclerosis is of importance in the etiology of chronic valvular disease. Among these causes are to be mentioned the general wear and tear incident to advancing years, excessive physical labor, overindulgence in food, alcohol and tobacco, and gout. Chronic interstitial nephritis is also frequently associated with primary chronic valvular disease. Whether the chronic valvulitis, the arterio-sclerosis and the nephritis are due to the same cause or one arises in consequence of the others, is not clear. It is certain, however, that two or all three of these conditions frequently occur together.

3. *Syphilis.*—Within the past decade our knowledge regarding the rôle played by syphilis in the causation of cardio-vascular disease has been greatly extended. We now know that a definite group of cardiac cases are unmistakably syphilitic in origin. With few exceptions the lesion thus far proved to be due to syphilis is aortic insufficiency. A few instances have been recorded in which the mitral valve was the site of a luetic infection. Doubtless in the near future it will be shown that all parts of the heart are more frequently invaded by the spirochæta pallida than is at present believed (see p. 689).

4. Occasionally a chronic valvular lesion is detected at birth. This may be due to congenital malformation or a fetal endocarditis. If a congenital anomaly is present the pulmonic valve is the one commonly at fault, the lesion being usually a stenosis associated with some other malformation of the heart. If an attack of fetal endocarditis has occurred, the valves on the right side of the heart are most commonly involved.

In rare instances the chronic endocarditis is due to a rupture of one of the valve segments. The aortic leaflets are those usually torn as the result of some severe muscular exertion.

Cabot has analyzed 600 cases of cardiac disease from an etiological standpoint. Ninety-three per cent. of these were classified in definite groups as follows:

Forty-five per cent. were rheumatic in origin. Of these 60 per cent. occurred in women, and 60 per cent. were in individuals under twenty-two years of age. *Twenty per cent. had a nephritic origin,* with an average age of thirty-six years. *Fifteen per cent. were arterio-sclerotic* in nature, with an average age of fifty-nine years. *Twelve per cent. were due to syphilis,* with an average age of forty-seven years, 70 per cent occurring in men. These cases were comprised of aortic insufficiency, aneurism, myocarditis; *five per cent. were due to goitre.*

Morbid Anatomy.—In acute endocarditis the physiological defect produced by the valvular lesion is a leakage due to the inability of the valves to properly close and also to an associated myocarditis. Obstruction to the blood current does not occur except in those instances of malignant endocarditis in which the size of the vegetations partially block the mitral or aortic rings. In the chronic form of endocarditis the lesion may cause obstruction or leakage or both.

When the lesion is secondary to simple acute endocarditis the ultimate result is a local fibrosis with some contraction of the involved area of the valve segment and a resulting deformity.

In the primary form of chronic endocarditis the valvular changes, particularly in the aortic cusps, are identical with those met with in arterio-sclerosis. The condition is non-inflammatory and is dependent on the same factors which lead to the arterial changes. These have been alluded to already in discussing the etiology.

The end result in both instances is essentially the same. The connective tissue, in common with all new connective tissue, tends to undergo contraction. This may curl the edges of the valve segments or produce shortening; in either case a leakage results. Or the edges of the cusps may undergo fusion along their opposed edges; later as they become stiffer and more rigid a stenosis is produced. As time goes on degenerative changes, calcification and atheromatous ulceration may develop or the chordæ tendineæ may undergo thickening and constriction and thus seriously interfere with the proper working of the valve.

In many cases of chronic endocarditis the cardiac damage is not limited to the valves. The lesions not infrequently extend to the adjacent areas. Thus in aortic lesions the first part of the aorta may be affected with sclerotic changes which also involve the orifices of the coronary arteries. In this way myocardial changes may be brought about through interference with the nutrition of the heart. When the mitral orifice is involved the inflammatory process may be spread into the heart muscle surrounding the ring. Owing to the close proximity of both the aortic and mitral valves to the auriculo-ventricular node and bundle, serious disturbances in the cardiac rhythm may occur if the inflammatory process invades the muscular tissue.

Combined Valvular Lesions.—The valvular defect may be single or multiple. Stenosis is often accompanied by some degree of leakage and conversely incompetence may be associated with some degree of narrowing. Furthermore, two or more valvular orifices are frequently affected. In children involvement of both the mitral and aortic orifices is not uncommon as the result of acute rheumatic fever. Aortic lesions are more commonly uncombined than mitral lesions. This is to be explained by the fact that syphilis is so frequently the etiological factor in aortic insufficiency. Secondary involvement of the tricuspid orifice is a common event in mitral disease.

Generally speaking two or more valvular defects are more serious than a single lesion but at times the ill effects of regurgitation may be offset by a certain degree of stenosis.

The morbid anatomy will be considered in greater detail when we take up the individual lesions.

Pathological Physiology of Chronic Valvular Lesions.—The normal heart performs its work with a regularity and precision which amply justifies Krehl's statement that it is the best motor known to man. "Sixty times a minute at least; 3600 times an hour; 86,400 times per day, for us heedful and heedless, does this shuttle of life flit to and fro; for us in tireless periods this pendulum of man's gravitation tells the seconds which will never return" (Allbutt).

Not only does it proceed smoothly and rhythmically in the performance of its work when the body is at complete rest but it also possesses the power of adapting itself to the varying demands made upon it.

Within reasonable limits the healthy heart rarely fails in any effort. This does not necessarily mean that every healthy heart is capable of performing the same amount of work or possesses the same power of meeting sudden emergencies. It is to be borne in mind that the heart muscle is subject to the same general laws as the skeletal muscles. The stronger and the better-nourished the general musculature, the more apt is the heart to be correspondingly strong, and the more capable of meeting any sudden demand that may be made upon it. This relationship which exists between the cardiac muscle and the skeletal muscles explains why the individual who has undergone a course of physical training or has become used to hard physical labor can take part in athletic contests or swing a heavy sledge hammer without producing exhaustion. On the other hand, the man who leads a sedentary life and whose muscles, as a consequence, become soft and flabby will certainly have a heart muscle correspondingly reduced in its capacity for meeting any unusual strain. Before such an individual can hope to do anything requiring exertion to which he is unaccustomed, the muscles must be exercised and hardened and this applies quite as much to the cardiac as to the voluntary muscles.

As a result of exercise the heart may become more or less hypertrophied without any injurious results; providing, of course, this is brought about gradually. That the strength of the heart has a great deal to do with feats of strength or endurance is borne out by the fact that the most successful athletes possess hearts of good size and a functional capacity above the ordinary It has long been known that race horses possess relatively large hearts.

The adaptability of the heart to cope with the various emergencies it is required to meet is also seen in the manner in which it overcomes these demands. "For example, when a ventricle in diastole becomes unusually filled (even up to six times its customary capacity), then with the next systole, it drives out not all the blood, perhaps, but at least several times the ordinary quantity. On the other hand, if the arterial resistance be suddenly increased, it is as promptly overcome by the succeeding ventricular contractions. There is no time lost in experimentation. The demand and the accomplishment occur together" (Krehl).

Were it not for this ability of the heart to adjust itself to changed conditions which may either gradually or suddenly thrust themselves upon it the individual who becomes the subject of a chronic valvular lesion would quickly succumb.

The heart valves perform two functions: (1) They direct the blood current in the proper direction. (2) They prevent leakage by tightly closing the openings. This is accomplished by the apposition of the valve segments which are controlled by the papillary muscles and chordæ tendineæ and also by the contraction of the ring of muscle surrounding the opening. During systole the orifices become smaller while during diastole they are relatively too large for the valves.

Now, when it comes to pass that the valves become deformed as the result of a chronic endocarditis, the heart is confronted with additional work. Either it must overcome the resistance caused by stenosis of one of the orifices or it must properly empty an abnormally distended cavity caused by leakage. Under normal conditions in a given interval, as much blood is driven out of the heart as flows into it. If, therefore, in the presence of a valvular defect, the heart were to work with the same energy as under normal conditions, an impairment of circulation would soon arise which would quickly lead to a fatal termination. Fortunately the heart is possessed of a reserve force which enables it to compensate for these defects and to so carry on its work that no circulatory ill effects are felt. When a heart is damaged by a valvular defect and at the same time does its work as efficiently as a normal heart, we speak of the lesion as a compensated one. The period of compensation is variable; it may last throughout life. In the majority of instances, however, the compensation is only relative and the patient will remain free from subjective symptoms only as long as care is taken to spare the heart in every way. Thus in some individuals varying amounts of exercise may be permissible while in others the circulatory equilibrium is maintained only when the patient is kept at complete rest. *Compensation* depends on three factors, namely: the extent of the valvular damage; the demands which may be made upon the heart either through exertion or through some acute intercurrent illness; and, lastly, the degree of hypertrophy which takes place. Hypertrophy of the heart, arising as the result of valvular disease, is variable in degree. It may be enormous as in cases of aortic insufficiency, or it may be very slight as in many cases of mitral insufficiency. This hypertrophy does not affect all the chambers of the heart uniformly but is more noticeable in those portions in which the valvular defect has led to increased functional activity.

In the majority of cases with a well-marked valvular lesion there comes a time when the heart begins to falter and is no longer able to accomplish the additional work demanded of it. The failure of the heart to carry on the burden may be due to the fact that the valvular defect gradually increases or it may be that the continued strain eventually leads to exhaustion of the cardiac muscle. In still other instances the heart is thrown out of its stride by some acute intercurrent illness which indirectly affects it. So long as the compensation is adequate, symptoms indicative of heart disease are absent. At first the symptoms may be trifling but they are apt to become worse either gradually or suddenly. When this condition of affairs arises it is spoken of as *failing* or *broken compensation* or *decompensation*.

Valvular defects of the heart are recognized clinically mainly by the presence of *murmurs* but it must not be assumed that the absence of a murmur is positive evidence that a valvular lesion is not present. Great widening of an orifice may be present and permit of the regurgitation of large quantities of blood without producing a murmur. In cases of stenosis of the mitral orifice it not infrequently happens that the murmur will disappear and yet a diagnosis of mitral stenosis can be made, with a fair degree of certainty, from the presence of other physical signs.

Regurgitant murmurs, as a rule, lack the rasping quality common to those associated with an obstructive lesion.

A soft blowing murmur may indicate that the valvular damage is slight or it may be significant of an enfeebled muscle. The distinction is usually readily made from a consideration of the associated signs and symptoms. Thus, if there are no symptoms or signs of beginning cardiac failure, one is safe in assuming that the valvular damage is slight; conversely, a feeble murmur associated with such symptoms indicates serious valvular damage or an associated myocardial degeneration.

A loud murmur is usually indicative of good compensation and points o a strong, healthy heart muscle. A change in the intensity of the urmur is very significant. If it is noted that a murmur is gradually ecoming less intense it is a fair assumption that the heart muscle is beinning to weaken and this is often the forerunner of failing compensaon. In such cases the murmur or murmurs may disappear entirely. , as a result of rest and cardiac medication, the circulatory equilibrium reëstablished the murmur begins to return, at first feebly, and as the eart tends to recovery the murmur becomes louder and louder. In ceptional instances the murmur is so loud that it may be heard at me distance from the chest wall; musical murmurs are not infrequently this character.

The duration of the murmur is also of importance. If it occupies t a portion of the systole or diastole the presumption is that the amage is slight while the entire replacement of the first or second sound y a murmur is indicative of an extensive lesion and one in which comnsation will be maintained with some difficulty.

It is of the utmost importance to bear in mind that the murmur is, r the most part, of relatively little consequence. This has been emhasized by Osler, who states that for those who are not adept in ausltation the best judgment of the condition of the heart may be gathered om inspection and palpation. With an apex beat in the normal tuation and a regular rhythm the auscultation phenomena may be ractically disregarded. The most important consideration in valvular isease is the condition of the heart muscle. So long as the lesion is ell compensated and the heart performs its work with practically the me efficiency as a normal heart there is nothing to be done except uard the patient against overexertion, or anything that will unduly crease the cardiac work. If, on the other hand, cardiac symptoms radually develop it is highly probable that the heart muscle is beginning o fail. The compensation may be broken in several ways. First, here may arise an exhaustion of the heart muscle which is brought about y continued overwork in the attempt to overcome the valvular defect r some extra strain may be imposed upon the heart which it is unable o meet. Second, the mechanism which governs the cardiac rhythm ay be disturbed and any of the abnormal rhythms may occur. Heart lock, auricular fibrillation and flutter are all apt to add to the strain of heart already taxed to its limit. The character of the various types f arrhythmia and the means employed for their recognition are disussed in full in Chap. XV. Finally, compensation may fail, as Stengel as pointed out, from extracardiac causes. This may be brought about y lack of regulated exercise, overeating, obesity, disturbances of the abominal circulation, improper breathing, thoracic diseases or advancing teriosclerosis with its attendant hypertension.

Another manifestation of decompensation is *acidosis* which results om failure to eliminate carbon dioxid from the blood. In many inances this can be recognized by the character of the breathing. The ly symptom that may be considered positively pathognomonic of idosis is hyperpnea or deep breathing, the so-called "air hunger" Kussmaul. This type of breathing is characterized by the amplitude the respirations and the distinct effort with which they are acmplished. They are heaving, the chest rises and falls with each respiration and often the accessory muscles of respiration are broug into play. The respiratory rate is not usually increased, and diffe markedly from the shallow, rapid respiration of pneumonia or t labored breathing of obstruction. It is pauseless and though the se- eral respirations may vary in depth so that a modified Cheyne-Stok respiration may be present, in general the excursions of the abdom and thorax are nearly the same with succeeding respirations. The is no cyanosis except that due to the cardiac disease. There may some drowsiness. Whenever possible the diagnosis of acidosis should confirmed by one of the simple laboratory tests (Howland and Marriot

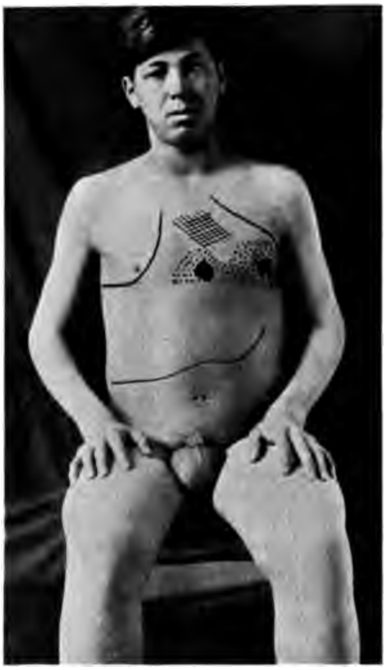

Fig. 399.—General edema occurring in broken compensation. Mitral and tricuspid insufficiency complicated by serofibrinous pericarditis. The cardiac dulness is enormously enlarged, especially to the left, the apex beat is displaced downward and outward. The scrotum and lower extremities are edematous, the hands and face cyanotic, the liver enlarged, tender and pulsating. The cross-hatched area indicates the location of the to-and-fro pericardial friction. The dotted regions indicate the areas and direction of transmission of the mitral and tricuspid systolic murmurs. The case is of rheumatic origin and the physical signs are shown as they occurred during the second attack of broken compensation.

Symptoms.—Irrespective of the valve involved or whether the lesion has produced obstruction, or leakage, or both, the symptoms which arise as the result of valvular disease are with a few minor exceptions essentially the same. In mitral lesions pulmonary congestion is a more marked feature than in the aortic cases and cyanosis is common. Children who are the subjects of chronic valvular disease are commonly anemic and undersized. In aortic insufficiency true angina or anginoid attacks of pain occur more frequently than in other forms of valvular disease, pallor of the face is common, and vertigo and mental symptoms are often very pronounced.

Shortness of breath is common to all forms of valvular disease and is present, at some stage of the trouble, in the great majority of cases. Prior to the stage of broken compensation it occurs only after some unusual exertion; the degree of exertion necessary to cause shortness of breath varies greatly in the individual case. When the heart fails, true dyspnea or orthopnea is common. In some instances patients are suddenly seized with paroxysmal attacks of dyspnea. These attacks are often not associated with exertion and not uncommonly occur at night. Cheyne-Stokes breathing often occurs in cardiac cases.

Aside from attacks of true angina or seizures resembling it, and which occur, for the most part, in aortic insufficiency, actual pain is not common as a result of valvular lesions. *Precordial distress* and a sense of substernal oppression are, however, quite common. Not infrequently the sense of oppression is referred to the epigastric region. Very often the precordial distress is associated with an annoying palpitation and throbbing. Chest pain in cardiac cases may be due also to a pulmonary infarct.

Edema is another very common manifestation of cardiac disease. It differs from the edema occurring in Bright's disease, in that it first manifests itself in the lower extremities and is much more noticeable in the evening after the patient has been up and about all day. After a night's rest the edema may disappear by morning. As the heart muscle becomes weaker, the edema not only persists but is apt to increase. The lower extremities become water-logged and in males the scrotum and penis may become tremendously edematous (Fig. 399). In extreme cases some one or all of the serous cavities may contain fluid. Hydrothorax is relatively common (see section on "Hydrothorax").

Gastro-intestinal symptoms are often marked. A not inconsiderable number of cases of failing compensation come to the hospital complaining of nausea and vomiting and it not infrequently happens that they have been treated outside for some stomach disorder. This can occur only as a result of carelessness as the most superficial examination will reveal the fact that the heart is at fault. This condition of affairs is brought about by tricuspid insufficiency which results in the damming back of the blood in the venous system, thus producing congestion of the liver and the gastro-intestinal mucosa. In extreme cases the liver becomes greatly engorged, very painful and may pulsate. Another condition sometimes produced by the hepatic congestion and gastro-intestinal catarrh is *jaundice*.

Cardiac cases quite commonly suffer from transient attacks of faintness and dizziness. Sleeplessness or sleep that is frequently broken is often a very annoying condition. In some patients, particularly those suffering from aortic insufficiency, hallucinations, delusions or a suicidal tendency are not infrequently encountered. Hemiplegia, or some other gross cerebral lesion, may occur as a result of an embolus.

Having reviewed the general aspects of valvular diseases we will now consider the individual lesions more in detail.

MITRAL INSUFFICIENCY

Etiology.—This is one of the most common, if not the commonest, of cardiac lesions. A leakage at the mitral orifice may arise under the following conditions: (1) The mitral insufficiency may be caused by an attack of acute endocarditis occurring in young people. In the majority of instances the endocarditis has resulted from an attack of acute rheumatic fever, chorea or tonsillitis. Although these conditions are the chief offenders, any one of the acute infectious diseases may produce the condition. I have already emphasized the fact that in the so-called rheumatic endocarditis it is the mitral valve which is principally involved. (2) In a second group composed, for the most part, of individuals who have passed the fifth or sixth decade of life, the leakage is caused by a chronic sclerosis of the valve. This is a part of a general arteriosclerosis and occurs without an antecedent history of acute endocarditis. Varying degrees of sclerosis of the mitral valve are encountered in nearly all people of advanced years and just as in the case of the arteries, it is indicative of the general wear and tear of work. (3) Occurring with even greater frequency is the leakage which occurs as the result of *relative insufficiency*. In this group the trouble is muscular and not

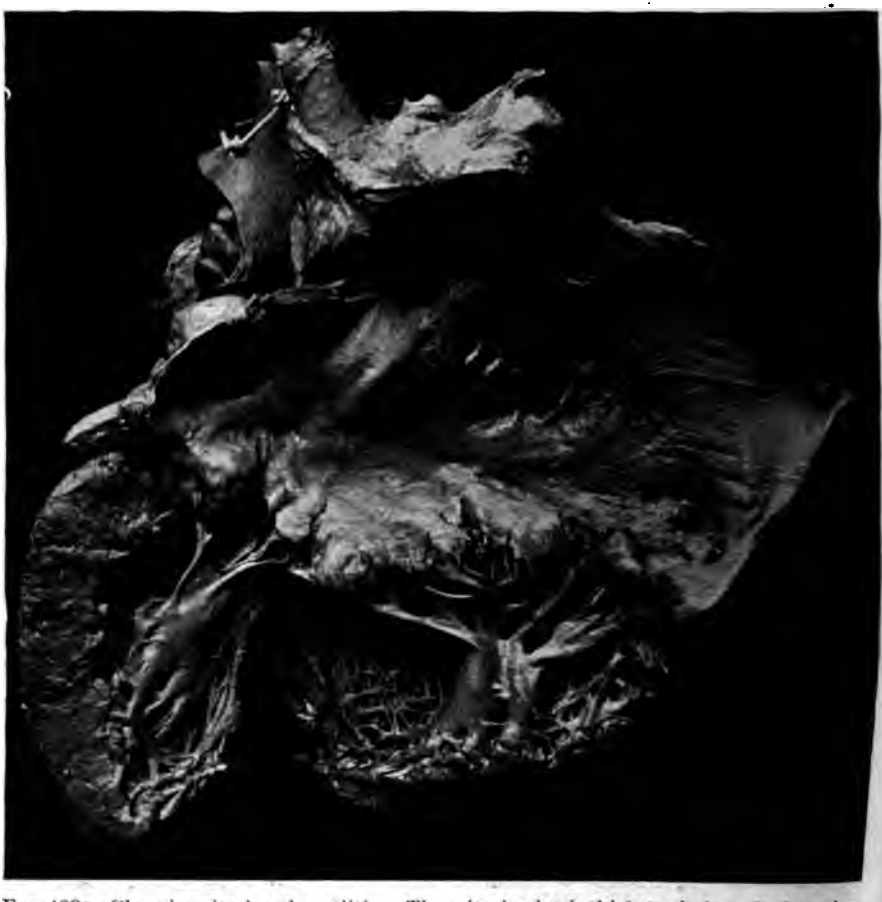

FIG. 400.—Chronic mitral endocarditis. The mitral valve is thickened along its free edges. the leaflets are slightly retracted, and the chordæ tendineæ are distinctly shortened.

alvular. The valve itself is healthy but owing to lack of tonicity or the result of acute or chronic myocarditis the left ventricle dilates d in so doing stretches the mitral ring. Thus while the valve under rmal conditions is capable of closing the auriculo-ventricular opening, aptation of its edges is prevented by the distended ring. Relative

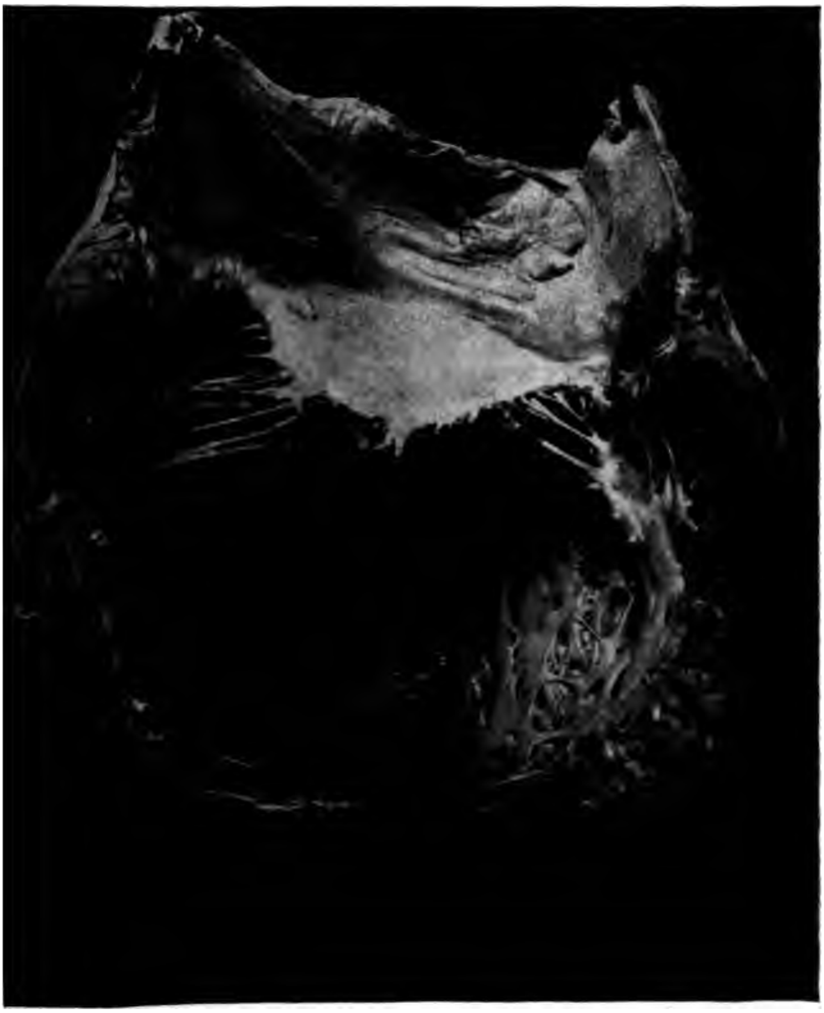

Fig. 401.—Mitral insufficiency. Showing very extensive tissue destruction. The ordæ tendineæ at the central part of the valvular margin have been destroyed by ulcera-n, leaving only short shriveled stumps. A few verrucose vegetations are seen. The ntricular wall is much thickened. (*Specimen from the Pennsylvania Hospital.*)

r muscular insufficiency may occur in a variety of conditions. It is frequent finding in the anemias, in the acute fevers, notably typhoid, d in conditions characterized by exhaustion such as Graves' disease, urasthenia, etc. Christian is of the opinion that stretching of the mitral ring as the result of chronic myocarditis, is the most frequent cause of the phenomena usually ascribed to an organic valvular lesion. In thirty-two out of forty-one cases of this type he found the mitral valve normal. Relative insufficiency of the mitral valve is also of common occurrence in other forms of valvulitis when compensation begins to fail. (4) Regurgitation may result from rupture of the valve or one of the chordæ tendineæ. This accident is very rare.

Morbid Anatomy.—In considering lesions involving the mitral valve it is to be borne in mind that neither the mitral nor tricuspid valve possesses distinct cusps as in the case of the aortic and pulmonic valves. Both the mitral and tricuspid valves consist of a veil or curtain which hangs in the ventricular cavity and is continuous around its whole circumference. Although these valves are commonly spoken of as having an anterior and a posterior leaflet, no such division actually exists.

When chronic endocarditis of the mitral valve succeeds the acute form of the disease, it is usually slow in forming. The newly formed connective tissue within the substance of the valve gradually shrinks, causing both a shortening of the valve and a curling up of its free edges (see Fig. 400). In other instances the free edges of the valve are drawn together, thus producing some narrowing of the orifice as well as insufficiency. Along the free edges of the valve the tissue may be cartilaginous in character and in addition the chordæ tendineæ are shortened, sclerotic and often fused together. While in most instances chronic rheumatic endocarditis is gradual in its evolution, there are instances in which the process is very rapid in its progress. In still other instances the acute stage is very destructive, causing widespread erosion in both the valve and the chordæ tendineæ and causing within a few days an extensive leak (see Fig. 401).

In the primary sclerotic type the process is identical with what we encounter in arterio-sclerosis. It is non-inflammatory in character and as time goes on the fibrous changes become more and more marked. Later, salts are deposited in the valve and in extreme cases both the valve and ring may form a rigid membrane. The chordæ tendineæ may be involved, also becoming both thickened and contracted. In some instances the contraction is so marked that the valves seem to be inserted upon the apices of the papillary muscles. Some degree of stenosis is commonly associated with the insufficiency.

Emboli sometimes occur in mitral insufficiency. They may arise from a thrombus in the left auricle or from fresh vegetations on the valve. Occasionally venous thromboses are met with.

Pathological Physiology.—When leakage occurs at the left auriculo-ventricular opening, a part of the contents of the left ventricle is thrown back into the auricle during systole. This increases the amount of blood the left auricle is forced to accommodate. As a result the auricle becomes dilated and hypertrophied; damming back of the blood takes place in the pulmonary circulation, which results in raising the blood-pressure and this in turn increases the work of the right ventricle. The increased work thrust upon the right ventricle causes it to hypertrophy and just as in the case of mitral stenosis, the circulatory equilibrium will be maintained just so long as the right ventricle can, without undue effort, overcome the resistance in the pulmonary circulation. If, however, the leakage at the left auriculo-ventricular orifice increases to a int where the right ventricle is no longer able to maintain the pressure the pulmonary circulation, compensation fails.

Some degree of hypertrophy and dilatation of the left ventricle always ist in cases of mitral insufficiency. During ventricular diastole the ood flows from the auricle into the ventricle with greater force than rmal owing to the increased pressure in the pulmonary veins. In dition the amount that flows into the ventricle must be greater than nor- il if the body is to receive the proper amount of blood. The increased essure with which the blood flows plus the increased amount both lead dilatation or an increase in the capacity of the left ventricle.

Hypertrophy is brought about by reason of the fact that the left ntricle during each systole is forced to expel a greater amount of blood in normal. The greater amount is driven into the aorta and a very ich smaller amount escapes back into the auricle. The amount that gurgitates will of course depend on the size of the leak and determine the iount of hypertrophy necessary to compensate for the damage.

As we have already pointed out, however, it is the right ventricle iich really controls the situation. Failure of compensation is brought out either by an increase of the mitral leak or from changes in the iscle of the right ventricle. When the right ventricle is no longer able carry on the burden relative insufficiency of the tricuspid orifice occurs, e right auricle becomes dilated, and the blood is dammed back into the ge venous trunks. This results in an engorgement of the entire venous stem which produces cyanosis, edema and passive congestion of the scera, notably the liver (see "Tricuspid Insufficiency," p. 743). In dition some forms of arrhythmia are very common.

Symptoms.—If the leak is small and the compensation is adequate, symptoms will be present even with moderate exertion. In other ses the compensation may be good unless some unusual strain is put upon e heart. In such cases undue exertion may produce shortness of breath d precordial pain or oppression. In long-standing cases in which the mpensation falls just short of being complete, shortness of breath curs on the slightest exertion, there is a bluish tint of the lips, the igers may be clubbed and recurring hydrothorax, usually on the right le, may occur. Patients suffering from chronic mitral disease are es- cially liable to attacks of bronchitis during the winter.

As time goes on and compensation gradually fails, the dyspnea and anosis become more and more marked, slight edema of the legs may noted at night and there are evidences of congestion of the lungs. The ilmonary changes are never as marked as in cases of mitral stenosis. ie tendency is rather to congestions and edema of the bases of the lungs ther than the chronic fibrous changes sometimes noted in association th obstruction of the mitral orifice. Hemoptysis, although not as quent as in cases of mitral stenosis, often occurs.

Compensation may fail gradually or it may be precipitated abruptly the result of some unusual exertion, mental shock or an intercurrent ute illness.

Broken compensation is characterized by distressing palpitation of the art, attacks of precordial oppression, extreme cyanosis and attacks dyspnea or orthopnea which may occur in paroxysms and result from lmonary congestion or edema. The patient is restless and sleep often broken accompanied by nightmare, or by attacks of cardiac oppression. For a more detailed description the reader is referred to the section on "Tricuspid Insufficiency."

Even a very severe attack of broken compensation may be recovered from and the patient may remain well for years, but as a rule, once the circulatory equilibrium is broken there is apt to be a recurrence of the attacks.

Physical Signs.—*Inspection.*—If compensation is well maintained, the patient presents the appearance of good health. Depending on the degree of failure of compensation there will be noted some cyanosis of the face, edema of the lower extremities and difficulty in breathing.

In children if the heart is much hypertrophied the cardiac region may be slightly bulged forward. The apex beat is displaced to the left and sometimes downward into the sixth interspace. In long-standing cases the apex beat may be as far to the left as the mid-axillary line. Extension of the cardiac impulse to the left is brought about partly by the enlarged left ventricle and partly by the hypertrophied right ventricle. The former tends to carry the apex downward while the latter pushes the apex outward to the left. In compensated cases the impulse is not diffuse and is much stronger than normally. If compensation fails, the cardiac impulse is very diffuse and undulatory in appearance.

Hypertrophy of the heart as the result of the mitral regurgitation is usually associated with the rheumatic group of cases. In elderly people suffering from the arterio-sclerotic type of the disease the apex beat may be in its normal position and hypertrophy of the left ventricle is very slight.

Marked pulsation in the epigastric region, due to hypertrophy of the right ventricle, is often marked. Unless the leak is very slight, some enlargement of the veins at the root of the neck is usually present. When compensation fails, these veins may become enormously enlarged and pulsate with each systole.

Palpation.—This serves to indicate the strength and character of the impulse. In not a few cases there is felt a systolic thrill which extends as far to the left as the axilla. The shock of the second sound may also be felt.

The impulse in mitral insufficiency is normal in well compensated cases and the blood-pressure is about normal. If there is an associated kidney lesion the pulse often feels hard and the blood-pressure may be distinctly higher than normal.

According to Broadbent it is a safe working hypothesis to assume that a mitral murmur is not attended with serious regurgitation while the pulse remains normal. While marked irregularity of the pulse is often the precursor of a fatal termination, irregularity of the heart's action may occur early and is not inconsistent with good compensation and fair health for a period of years.

The irregularity of the pulse in mitral disease is generally due to *auricular fibrillation*, and is very characteristic (see p. 730). The varying size of the pulse waves is extremely striking. The ventricular contractions remain coördinate, but their rhythm is disturbed and the beats are very irregular, being now large and now small, although the pulse periods are nearly equal (see Figs. 143 and 153).

Percussion.—The area of cardiac dulness is increased and this in-
se may in extreme cases be tremendous. Not only is the left border
ardiac dulness extended far beyond the normal limits, but it also may
nd to or beyond the right parasternal line. The upper limit may be
he second rib (see Fig. 172).

Auscultation.—The recognition of mitral insufficiency depends largely
he presence of a systolic murmur heard at or above the apex and trans-
ted toward the axilla. The sound produced by the backward escape
he blood is subject to many variations. There may be nothing
e than a roughening of the first sound. If an actual murmur is present,
ay be rasping and loud with, at times, a musical quality. In other
ances it is soft and low-pitched. The murmur may be of short dura-
or it may occupy the entire systolic period. A noticeable feature
he murmur is that it diminishes in intensity in contrast to the cres-
lo murmur of mitral stenosis. The murmur may be transmitted
a short distance to the left, but in well-marked cases the sound is
ly always propagated to the axilla and is often heard in the left inter-
ular region (see Figs. 199 and 200). When loud, it may be heard over
entire chest, and in such cases the question often arises as to whether
murmur is single and has its origin at the mitral or aortic orifice
hether there is a double murmur present. If the murmur at the
diminishes in intensity as the base of the heart is approached but
the same duration and tone, it is caused by mitral insufficiency
e. If, however, the murmur diminishes and then increases in in-
ity as the aortic area is reached and in addition is of shorter or
er duration and differs in tone, there is a strong probability that two
inct murmurs are present. In cases of mitral insufficiency the sec-
pulmonic sound is sharply accentuated and often reduplicated.

enerally speaking, a loud murmur implies strength of contraction
indicates good compensation. On the other hand, a short, faint
mur which varies in intensity and duration in successive beats
cates a weak muscle. If in a case under constant observation it
ted that the murmur is gradually becoming less intense, one should
uspicious of failing compensation. And conversely the first evidence
ting to restored compensation may be the return of the murmur
he gradual increase in intensity of a previously feeble murmur.

With an actual leak at the mitral orifice it is not always easy to deter-
e whether the insufficiency is due to an organic lesion of the valves
o relative insufficiency due to myocarditis, the anemias or some toxe-
. As a rule the *murmur of relative insufficiency* is less intense and
pt to disappear or become more marked with change of posture.
s it may be heard in the recumbent and not in the erect position.
thermore, if the murmur is associated with anemia or some acute
ction, the murmur disappears as the general condition improves.
Oille, Graham and Detweiller[1] have expressed the belief that *pul-
ary systolic murmurs* so frequently heard in "run down" and
mic individuals are not functional but are indicative, in many in-
ces, of mitral regurgitation.

A not infrequent source of error is the presence of a *cardio-respiratory
mur.* This murmur is relatively common and, while heard more often
more distinctly about the apex and to the left of the heart, should

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[1] *Jour. Am. Med. Assoc.*, October 2, 1915.

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after the patient climbs a flight of steps rapidly or hops on one foot fifty or sixty times.

There can be no doubt that murmurs indicative of mitral valve disease are less frequent than was formerly believed to be the case. Balfour, Christian and others are of the opinion that if the valve is sufficiently diseased to produce incompetence there is also some stenosis present. If this be true the only sure proof of disease of the mitral valve is the presence of some one of the signs of mitral stenosis.

MITRAL STENOSIS

In estimating the relative frequency of the different valvular defects mitral regurgitation is usually given first place. Most observers place mitral stenosis and aortic regurgitation second. MacKenzie, on the other hand, has expressed the belief that mitral stenosis is the most common of valvular defects with which heart failure is associated.

Etiology.—This lesion is encountered more frequently among females than males. This may be ascribed to the fact that acute rheumatic fever, chorea and tonsillitis prevail more in girls than in boys and also because in both these diseases the endocarditis more often affects the mitral valve. Acute rheumatic fever is credited with being the etiological factor in from one-half to two-thirds of the cases. In not a few cases of mitral stenosis it is not possible to obtain a definite history of any etiological factor. In the great majority of cases, however, the stenosis is the direct consequence of one or more attacks of acute rheumatic endocarditis. Very often the stenosis develops in chronic endocarditis as a sequel to a previous insufficiency. In such cases the valve becomes more and more sclerotic until finally the signs of obstruction overshadow those of regurgitation. Chronic interstitial nephritis is not uncommonly associated with this type of the disease, the valvular lesion having apparently an arterio-sclerotic basis. A few cases have been reported in which the stenosis apparently followed a severe blow on the chest. Experimentally hemorrhagic extravasation into the pericardium and endocardium can be produced by blows inflicted upon the precordium.

Morbid Anatomy.—As the result of an attack of acute endocarditis, changes are produced in the valve itself, in the chordæ tendineæ, or in both. This lesion is never recognized during the course of the acute process which produces it. It is only later when cicatrization is established and narrowing of the mitral orifice is produced that the characteristic features of the condition appear. While in many cases the initial damage does not spread, the tendency of the scar tissue is to extend, thus making the valve defect progressively worse. The initial stage is similar to that seen in mitral insufficiency. When the free edges of the valvular curtain are chiefly concerned, the deformity usually assumes the well-known funnel-shaped orifice (see Fig. 402). This is the type of defect which is generally seen in early life as the result of a rheumatic infection. On the other hand, the "button-hole" form of stenosis is observed with far greater frequency in adults (see Fig. 202). In this type the whole valve becomes thickened as the result of an overgrowth of fibrous tissue. This scar tissue eventually leads to puckering and thickening so that the valve is converted into a transverse septum or diaphragm with a narrow slit between the auricle and ventricle. The opening may be still

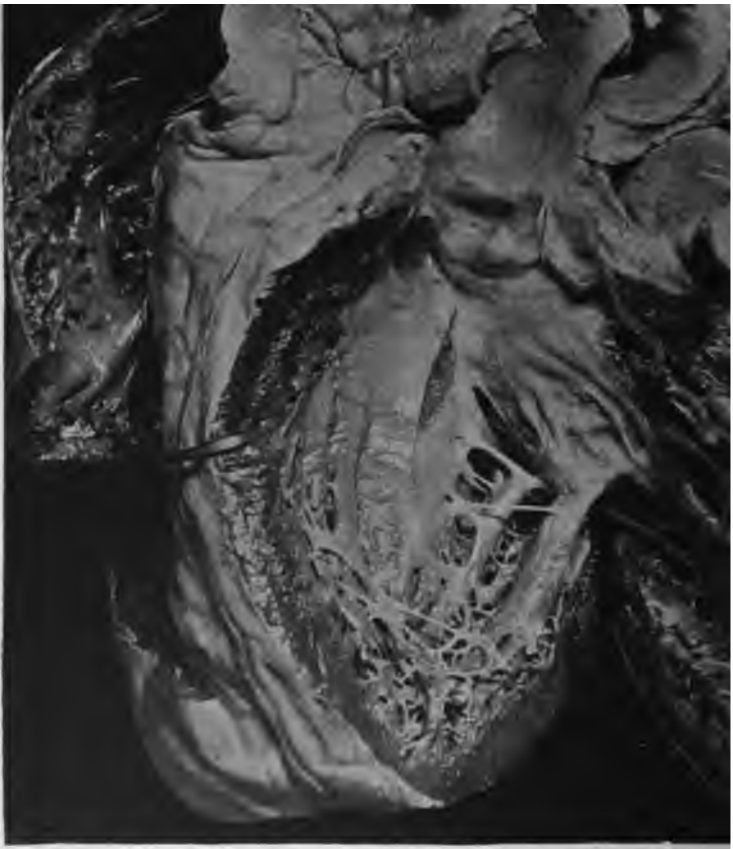

FIG. 402.—Mitral stenosis. Mitral orifice was not incised; its leaflets

There is some difference of opinion as to whether pure stenosis without insufficiency ever occurs. In view of the character of the lesion it can easily be understood, however, that some leakage is inevitable in many of the cases, although the clinical evidence of this may be masked by the more pronounced signs of the stenosis.

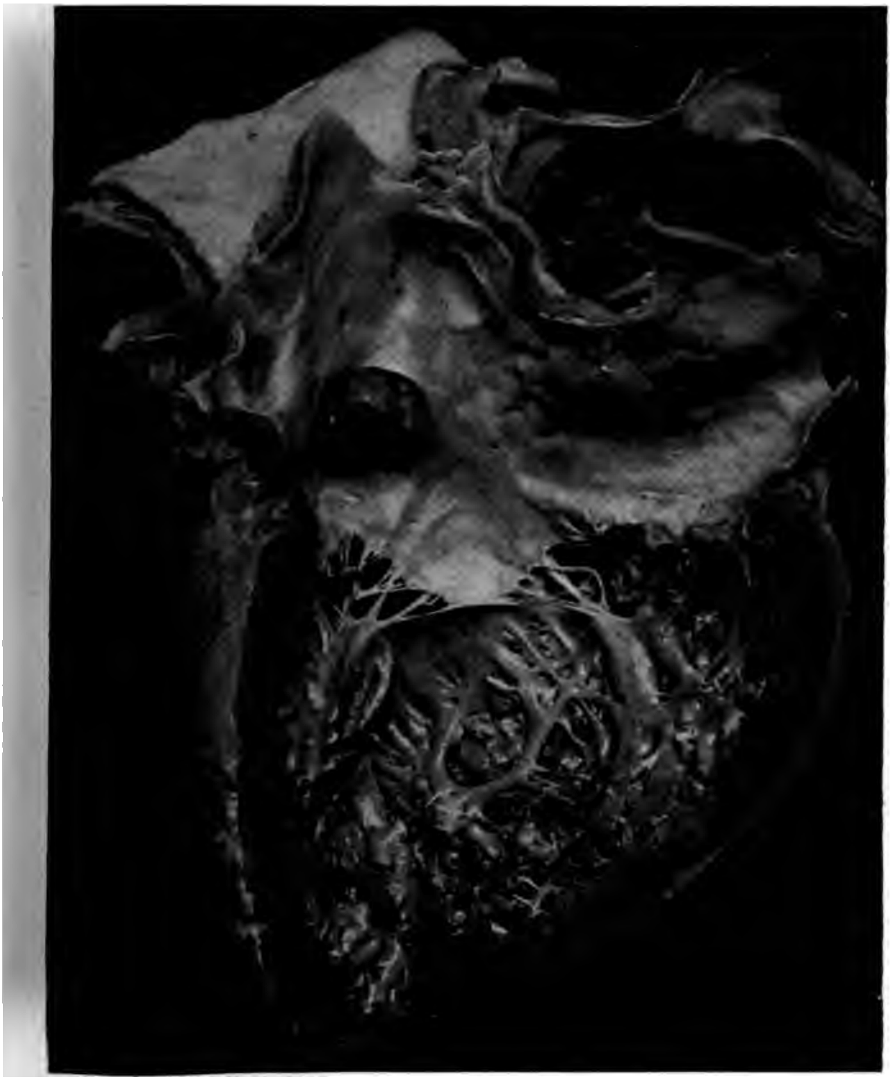

Fig. 403.—Ball thrombus in the left auricle. A large, dark, ball-shaped thrombus lies free in the left auricular cavity. Numerous smaller mural thrombi are seen enmeshed in the columnæ carneæ and chordæ tendineæ. The left ventricular wall, which is elsewhere hypertrophied, is noticeably thinned near the apex. (*Photograph by Dr. Alfred R. Allen.*)

Although there is usually some evidence of hypertrophy of the left ventricle it is not apt to be marked unless a considerable degree of insufficiency is present also. The chamber which bears the brunt of the attack is the left auricle which may become enormously distended. Backward pressure in the pulmonary circulation leads to pulmonary congestion and finally to hypertrophy and dilatation of the right ventricle. Thrombi, free or attached to the auricular wall, may form as the result of stasis, and from these emboli may be swept off, thus adding to the gravity of the situation (see Fig. 403).

Pathological Physiology.—Although the ultimate results are similar when compensation fails, the problem of maintaining the circulatory equilibrium in mitral stenosis is somewhat different from that in cases of insufficiency. In the former the difficulty lies in filling the left ventricle and, from the very beginning, in overcoming the stasis in the pulmonary circulation; in the latter the important factor is the emptying of the left ventricle and it is not until the leakage assumes serious proportions that disturbance in the pulmonary circulation becomes a menace.

The effect of obstruction at the mitral orifice first manifests itself in the left auricle, the wall of which becomes slightly hypertrophied. Inasmuch as the capability of the left auricle to hypertrophy is limited. this chamber sooner or later dilates. (See Fig. 198). Even prior to this it is unable to entirely empty itself and as a result the blood is dammed back into the pulmonary circulation. This in turn increases the work of the right ventricle which hypertrophies in order to overcome the increased resistance in the pulmonary vessels. Just as in mitral insufficiency the key to the situation is the ability of the right ventricle to overcome the resistance in the pulmonary circulation. Forced to maintain the pressure in the pulmonary vessels, the right ventricle dilates and this in turn eventually leads to dilatation of the right auricle with damming back of the blood in the systemic veins as the result of tricuspid insufficiency. In mitral stenosis leakage from the veins into the peritoneal cavity is often a marked feature. This results from secondary, relative, tricuspid insufficiency. The failure of the right ventricle also leads to stasis in the pulmonary circulation which manifests itself by the symptoms and signs of hydrothorax or passive congestion. In long-standing cases the stasis of the blood in the pulmonary vessels may lead to the deposition of blood pigment. As a result, the lung becomes of a reddish-brown color. In addition, there is an overgrowth of fibrous tissue imparting to the lung a tough, inelastic feel. The condition is known as *brown induration of the lung.*

An occasional effect of distention of the left auricle is pressure on the *left recurrent laryngeal nerve.* Fetterolf and Norris[1] have collected 37 instances. Their explanation is, that such distention by dilatation of, and upward pressure on, the left pulmonary artery may cause left recurrent laryngeal paralysis by compression of this nerve between the last-named vessel and the aortic arch.

Symptoms.—The fact that mitral stenosis is so frequently the forerunner of serious cardiac failure has given the impression that it is always followed by evil results. Quite the contrary is the case, however. There are many individuals in whom mitral stenosis exists in a latent form and in whom the discovery of its presence is made accidentally. On the other hand, a well-marked mitral stenosis is often the cause of cardiac symptoms which are ascribed to other lesions. In a study of 200 autopsied cases of mitral stenosis Cabot[1] states that the condition was overlooked in 53 per cent.

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[1] *Am. Jour. Med. Sc.*, May, 1911.

A patient with well-marked physical signs may be entirely free from symptoms or at the most suffer from slight dyspnea on exertion. Breathlessness is perhaps the earliest manifestation and with it there is often a slight cyanotic tinge of the lips. An appearance of ruddy health is often present, due to the congestion of the fine capillaries over the cheek bones. When the lesion develops in adults it is often well borne for an indefinite period. In the type which arises as the result of an attack of acute rheumatic fever in children the symptoms are usually marked and become progressively worse. Children subject to this lesion are often delicate looking, undersized, and are especially liable to secondary attacks of endocarditis (see Fig. 15).

Pulmonary attacks may develop before compensation is seriously interfered with. Thus as the result of fibroid changes in the lungs (brown induration) the patient may have cough and expectoration. The sputum at this stage of the trouble may be blood-streaked from time to time. Under the circumstances the blood is the result of the high pressure in the pulmonic vessels which causes the rupture of the capillaries. Individuals suffering from mitral stenosis are extremely liable to attacks of bronchitis at which time blood-streaked sputum is especially apt to occur. What has been described as a *pseudo-tuberculous type* of mitral stenosis is not infrequently seen in young women. Such individuals are pale, underweight, and are subject to frequent attacks of bronchitis which is often attended with blood-streaked sputum, especially at the menstrual periods. When compensation fails, bloody sputum or even small hemoptyses very commonly occur as the result of pulmonary infarcts. In a few cases the voice is husky or the cough is brassy in character as the result of paralysis of the left recurrent laryngeal nerve.

Precordial pain and tenderness are often complained of. Riesman states that in young girls complaining of precordial pain, the possibility of mitral stenosis should always be borne in mind. It is a common lesion, especially among the Russian Jewish population, and is often overlooked. Pain may be present also in the epigastrium as the result of engorgement of the liver. A sense of oppression in the precordium and attacks of tachycardia or palpitation are often annoying symptoms. Embolic phenomena are encountered more often in this form of valvular disease than any other. The emboli may arise from a secondary vegetative endocarditis but are more often due to thrombi in the left auricle or pulmonary vessels. Pulmonary infarcts are common. A not infrequent accident is the occurrence of hemiplegia due to an embolism; and this may be the first indication of the presence of the cardiac lesion.

When compensation finally fails, the evidences of venous stasis become marked. There is no essential difference between the phenomena which arise as the result of failing compensation in mitral stenosis and those occurring in mitral insufficiency, save in one particular. In the latter the venous stasis results in marked subcutaneous edema while in mitral stenosis an excessive amount of peritoneal fluid may appear before edema of the legs and may persist after the latter disappears. In addition engorgement of the liver is more evident in cases of obstruction. In many of the fatal cases fever and other evidences of an acute infection are frequently present.

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[1] *Trans. Assoc. Am. Phys.*, 1914.

Physical Signs.—*Inspection* may show nothing abnormal. In children the precordium may bulge forward and marked pulsation may be seen in the interspaces to the left of the sternum. In adults the apex beat may occupy its normal position or be displaced slightly to the left. This may be due to hypertrophy of the left ventricle, or to dilatation and hypertrophy of the right ventricle. In the later stages of the disease, when dilatation of the heart has taken place, the cardiac impulse may be wavy and diffuse. Pulsation may be seen to the right of the sternum and also in the epigastrium. In cases in which hoarseness is present, direct inspection of the larynx will usually show paralysis of the left vocal cord.

Palpation.—In many cases of mitral stenosis during the stage of compensation, the diagnosis may be made by palpation alone. This is possible through the detection of a *presystolic thrill* felt slightly above and to the right of the apex beat. If the hand is placed over this portion of the precordium, there is imparted to it a fine vibration which occurs during all or a portion of the diastole and terminates in the abrupt, sharp shock of the first sound. The thrill may not be present constantly in the same case. In some instances it is best felt during expiration; in others, it is brought out by slight exertion; while in still other cases it may be detected by altering the posture of the patient. The presystolic thrill may precede the appearance of the presystolic murmur by several years (Mackenzie). In addition to feeling the shock of the first sound at the apex there may be present also a distinct shock accompanying the second sound at the pulmonic area.

In the later stages of the disease when the compensation becomes broken, the thrill usually disappears. A presystolic thrill may occur also in association with Flint's murmur. The differentiation of Flint's murmur from that of the mitral stenosis will be considered under "Diagnosis."

Palpation confirms inspection as regards the character of the cardiac impulse.

The *pulse* at the wrist is usually small. The blood-pressure as a rule varies but slightly from the normal, but the pulse pressure is often diminished. The most interesting feature connected with the pulse is the irregularity which develops during the later stages of the disease and, in some instances, when compensation is well maintained.

Auricular fibrillation, of all the types of arrhythmia, is by far the most common. While it may occur in any form of heart disease, it is encountered most frequently in mitral disease and especially in cases of mitral stenosis. It is estimated that about seventy per cent. of patients exhibiting symptoms of myocardial degeneration and circulatory failure in hospital practice, are the subjects of this derangement. At one time auricular fibrillation was often referred to as pulsus irregularis perpetuus. It is now recognized that the condition is not always permanent; it may occur transiently during convalescence from one of the acute fevers or it may appear in a chronically damaged heart and disappear either temporarily or permanently. In the majority of cases, however, the irregularity once it appears, persists. The condition is readily detected, in all but a few insances, by the ordinary clinical methods and it is rarely necessary to resort to instrumental methods to determine its presence.

Auricular fibrillation is characterized by a pulse which is wholly irregular in character with beats varying both as to sequence and force (see Figs. 143 and 153). It will be noted also in many cases that there is a marked discrepancy between the number of beats counted at the wrist and those heard at the apex of the heart. Thus on auscultation the heart rate may be 150 beats per minute while the rate as felt at the wrist may be only 100. This finding is referred to as pulse deficit, and is occasioned by the failure of many weak ventricular contractions to produce a palpable pulse wave at the wrist. This form of cardiac arrhythmia is to be distinguished from all other arrhythmias in that, with an increase in rate, the disordered action becomes more evident. As the condition is dependent on degenerative changes in the auricle a further result of failure of the auricle is the disappearance of presystolic murmurs with the onset of the fibrillation.

In not a few cases auricular fibrillation is combined with partial heart block. This is apt to be the case if the degeneration of the heart muscle is general and involves the bundle of His. When this is the case the irregularity will be less evident in rates of from sixty to seventy per minute than in those cases reaching twice these numbers. In extreme cases complete heart block may occur.

A less frequent manifestation of auricular failure is *auricular flutter.* This condition is characterized by extreme rapidity of the auricular contractions, the rate ranging from 200 to 400. The ventricular rate is generally one-half the auricular rate but any degree of block may be present.

Percussion.—In the region of the apex, cardiac dulness may extend farther to the left than normal in case there is much hypertrophy of the left ventricle. At the base, dulness is apt to extend higher and farther to the left than is ordinarily the case owing to dilatation of the conus arteriosus; dilatation of the pulmonary artery may be a factor also. Usually cardiac dulness extends slightly beyond the right border of the sternum, as the right auricle is hypertrophied or dilated. When compensation fails, dulness to the right of the sternum may be marked. Enlargement of the cardiac dulness upward and to the left is due to dilatation of the left auricular appendage. This rarely if ever occurs in simple mitral insufficiency (see Fig. 183).

Auscultation.—The auscultatory signs in mitral stenosis are often extremely perplexing, especially to the beginner. In many cases we have three sounds to deal with instead of two, as in other valvular lesions. In addition the character of the murmur varies according to the stage of the disease. It is important to bear in mind that the lesion which leads to stenosis is a progressive one and for this reason both the signs and the symptoms will vary in different cases and in the same case from time to time.

There are several murmurs occurring in cases of mitral stenosis. With one exception they are heard over a small, circumscribed area a little to the right of the apex beat and have a very limited range of transmission. The most characteristic sign of mitral stenosis, and the first of the murmurs to appear, is the *presystolic murmur.* This murmur is sharply localized, the *punctum maximum* being at or a little to the right of the apex beat (see Fig. 201). It occurs late in the diastolic period and is rumbling, rough or vibratory in character. It is usually of brief duration and precedes or runs up to the first sound. In some instances the duration is relatively long and may occupy a greater portion of the diastolic period. Another distinctive feature of the presystolic murmur is that it is crescendo in character. The pitch and intensity of the sound rises rapidly and abruptly terminates with the loud snap of the first sound, although occasionally there is a brief interval between the cessation of the murmur and the first sound. The murmur is often accompanied by a presystolic thrill. The presystolic murmur is usually present in well-compensated cases of mitral stenosis. Its genesis has already been discussed. In the later stages of the disease, when the auricle becomes paralyzed and auricular fibrillation sets in, the murmur disappears.

Of almost equal importance is the character of the *first sound.* This occurs immediately after the presystolic murmur. It is very loud, of short duration, sharply accentuated and closely resembles an accentuated second sound. It is, indeed, often mistaken for the later, especially by the beginner. The third factor is the second sound at the apex. Thus we have occurring in close succession the presystolic murmur, the accentuated first sound and the normal second sound. It is to be remembered, however, that the second sound at the pulmonic area is usually accentuated as the result of the increased pressure in the pulmonary vessels and in addition it may be reduplicated.

As the stenosis increases another murmur, *diastolic* in time, appears. According to Mackenzie this murmur is limited to the area about the apex. It begins immediately after the second sound and while at first faint and inconstant it gradually becomes more pronounced. This murmur at first occupies the first portion of the diastolic period and is diminuendo in character. It may, however, merge into the presystolic, thus producing a continuous murmur throughout diastole. It is loud in the beginning, then diminishes in intensity and finally ends in the crescendo presystolic portion. With failure of the left auricle the presystolic portion disappears and the diastolic portion may remain, occupying a portion or all of diastole. This diastolic murmur is produced by the accumulation of blood in the auricle during the ventricular systole. It begins with the opening of the mitral valves at a time when the ventricular pressure is below that of the auricle.

Another diastolic murmur commonly heard in cases of mitral stenosis is one which is heard along the left border of the sternum. This murmur, sometimes known as the *Graham Steell murmur,* is ascribed to *incompetence of the pulmonary valves* as the result of the high pressure in the pulmonary artery. It closely resembles the soft blowing murmur associated with aortic regurgitation. Goodman[1] heard this murmur 12 times in 24 cases of pure mitral stenosis and 5 times in 12 cases of combined mitral stenosis and regurgitation. Hemoptysis seemed to be more frequent in cases associated with this murmur than otherwise. In 22 out of 50 autopsied cases in which this murmur was present Cabot[2] was unable to demonstrate disease of the pulmonary valves, dilatation of the pulmonary artery or any evidence of aortic incompetence.

Finally there may be associated with these presystolic and diastolic murmurs a short, soft, *systolic murmur* at the apex. This murmur is probably due, in most instances, to the presence of slight incompetence. Mackenzie, however, states that it differs from the murmur of true regurgitation in that it begins a short interval after the first sound, rises in intensity and terminates in the second sound.

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[1] *Am. Jour. Med. Sci.,* Feb., 1919.

[2] *Trans. Assoc. Am. Phys.,* 1914.

The untangling of the numerous sounds heard in the region of the apex in cases of mitral stenosis is usually a perplexing task for the beginner. Inasmuch as the auscultatory phenomena are confined to the region of the apex, it is usually possible to find a point at the base of the heart where the two cardiac sounds are unmistakable. By gradually approaching the apex, it can usually be determined in what respect they change or whether additional sounds develop.

It has already been pointed out that the lesion of mitral stenosis is a progressive one and that for this reason the signs and symptoms show corresponding changes. Broadbent has made a useful clinical division to indicate these changes. He recognizes *three stages:* (1) That of good compensation in which the presystolic murmur, the snapping first sound and the second sound are plainly audible at the apex. A diastolic murmur may appear in this period. (2) The presystolic murmur persists, but is associated with a diastolic murmur, the first sound is even more accentuated, and, of extreme diagnostic importance, the second sound at the apex disappears. It is in this stage that errors are most apt to occur, as the long continuous diastolic and presystolic murmur and the accentuated first sound may be mistaken for a systolic murmur and accentuated second sound. Dyspnea, hemoptysis and other evidences of stasis in the pulmonary circulation are often present in this stage. (3) The presystolic murmur and thrill have disappeared but a short diastolic murmur may persist. The second sound is inaudible and all that may remain of the auscultatory signs is the sharply accentuated first sound. Auricular fibrillation, as manifested by marked irregularity of the pulse and more or less disappearance of the murmurs, is common during this stage. The evidences of stasis in the pulmonary circulation become increasingly evident. To these three stages Hirschfelder has added a fourth in which there are present all the manifestations of broken compensation.

Diagnosis.—The diagnosis of mitral stenosis in the stage of compensation offers no serious difficulties, as the presence of the presystolic thrill and murmur and the snapping first sound are quite distinctive. The real difficulty occurs in the second and especially the third stage, when the characteristic auscultatory features have disappeared. In such cases the snapping first sound, and the presence of auricular fibrillation, even in the absence of cardiac murmurs, should arouse one's suspicions that the cause of the cardiac failure is mitral stenosis. Patients admitted to hospitals in the last stages of broken compensation offer the greatest diagnostic difficulty.

Flint's Murmur.—The association of this murmur with aortic incompetence and the mechanism of its production have been considered (see pp. 250 and 738). Like the murmur of mitral obstruction it is presystolic in time and heard at or near the apex of the heart. Thayer[1] found Flint's murmur present in 33 of 58 cases of aortic insufficiency and Phear[2] has reported 46 cases in which there was a presystolic apical murmur without mitral stenosis; in 17 instances there was aortic incompetence; in 20 adherent pericardium; in the remainder there was no valve lesion but in some of

[1] *Trans. Assoc. Am. Phys.*, 1901.

[2] *Lancet*, 1895, ii, 716.

these there was dilatation of the left ventricle. The Flint murmur is distinguished from the presystolic murmur due to mitral stenosis by the following points: It is never as loud and rasping in character and is not associated with the accentuated first sound. In mitral stenosis the systolic shock of the first sound can be felt while in the case of Flint's murmur the cardiac impulse is diffuse and heaving. In addition Flint's murmur is associated with the arterial phenomena seen in aortic regurgitation.

Tuberculosis.—Strange as it may seem a diagnosis of tuberculosis instead of mitral obstruction is not uncommon. As a rule it is to be ascribed to carelessness. Although there are cough, expectoration, at times blood-streaked sputum, sometimes fever, a rapid pulse and the presence of râles in the lungs in both conditions, careful examination of the heart will usually give the correct clue as to the origin of the symptoms. Among 2113 cases seen at the Phipps Institute there occurred 13 instances of mitral stenosis and 9 more in which there was a double mitral lesion. Stenosis of the mitral orifice and pulmonary tuberculosis may occur in the same individual.

Aneurism.—Reference has been made to the fact that in some instances distention of the left auricle may cause *paralysis of the left recurrent laryngeal nerve.* In such cases there is hoarseness, a brassy cough, and paralysis of the left vocal cord. Under these circumstances a diagnosis of aneurism of the arch of the aorta may be made. The auscultatory phenomena at the apex should serve to distinguish them. In case there is any doubt, the X-rays will easily differentiate the two conditions.

AORTIC INSUFFICIENCY

Clinically this is the most interesting of the valvular lesions. It causes very distinct alterations in the physics of the circulation and, even when combined with other valvular lesions, dominates the picture.

Etiology.—This lesion is encountered most frequently in males of middle age or those approaching this period. There may be an antecedent history of acute rheumatic fever, syphilis, pneumonia, severe exertion or alcoholism. Although rheumatic fever is a frequent cause it does not attack the aortic leaflets with anything like the frequency that it does the mitral valve. Rheumatic endocarditis affecting the aortic valve is usually seen in children (see Fig. 194). The importance of *syphilis* is becoming more and more apparent and a very large number of the cases seen among those in young adult life are due to this cause (see Fig. 190). A considerable number of infants dying soon after birth also have been found to have spirochetæ in the aorta. The part played by syphilis in the production of changes involving the first part of the aorta and the aortic ring has been fully discussed elsewhere (see p. 776).

Muscular strain is a well-recognized factor. In not a few cases there is no obvious cause, the lesion being a part of a general sclerotic process such as occurs in those of advancing years. In rare instances aortic insufficiency is due to a rupture of one of the valve segments as the result of severe muscular exertion or it may be due to a congenital deformity.

Morbid Anatomy.—The scar tissue which develops in the valve segments, whether it is produced by an acute inflammatory process, syphilis or a chronic sclerotic change, tends to shrink and produce deformity. The free edges of the segments may be curled back or as a result of retraction the free edges fail to come into complete apposition and thus fail to close the orifice. Sometimes one of the segments becomes perforated during the acute stage or a rupture occurs later as the result of strain. As a consequence of the leak thus produced, a part of the blood regurgitates from the aorta back into the left ventricle during diastole. The amount of blood which escapes backward will depend on the size of the opening left by the improper closure of the valves and by the tonus of the heart muscle.

If the opening is instantly made as the result of rupture of one of the segments a fatal result may be produced as the heart cannot accommodate itself to the sudden strain. In the usual type of case the opening is gradually formed and the heart is able to adapt itself to the increased work demanded of it. The chief burden falls upon the left ventricle which often becomes enormously hypertrophied. The heart of aortic insufficiency is one of the largest met with and is often referred to as the *cor bovinum* (see Figs. 181 and 193). Great hypertrophy of the heart, however, is not a constant accompaniment of aortic insufficiency as the opening may be very slight and the amount of blood which regurgitates small.

Relative insufficiency of the aortic valve rarely occurs as the result of cardiac dilatation but a permanent stretching of the aortic ring is not infrequent secondarily to syphilitic aortitis. In this case the valve segments themselves are intact but the leakage is produced by the inability of the valve to close the enlarged opening.

Disease of the first part of the aorta is also apt to involve the orifices of the coronary arteries and thus cause attacks of true angina. Even without involvement of the coronary openings the circulation in the coronary arteries may be embarrassed as a result of the lowering of the pressure in the aorta due to the backward leak.

In most cases of aortic insufficiency there is an associated arteriosclerosis and the systolic blood-pressure is high.

Pathological Physiology.—Failure of the aortic valves to properly close the aortic opening results in the back flow or regurgitation of a portion of the blood which has been thrown into the systemic arteries. The problem which the left ventricle faces is to expel during each systole the normal amount of blood which it receives from the left auricle plus the amount which regurgitates from the aorta. This naturally increases its work and results in hypertrophy. The latter is proportional to the amount of regurgitated blood. In cases which are recognizable clinically the hypertrophy is usually of an extreme grade. Another change produced in the left ventricle, as the result of the regurgitation from the aorta, is dilatation. This is brought about by the increased amount of blood it is forced to accommodate during diastole. Just as in the case of hypertrophy the degree of dilatation is dependent on the amount of the regurgitant flow from the aorta. In spite of the excessive amount of blood the left ventricle must expel at each systole, it empties itself in about the same length of time as the normal ventricle, although it does not always do so as completely. Whereas in aortic stenosis the left ventricle requires an increased amount of time to empty itself, a shortening of the systole is an advantage in aortic insufficiency, as it then has less blood to handle by reason of the curtailment of the diastolic period.

As the result of the great hypertrophy of the left ventricle the blood is driven into the aorta with tremendous force. This produces a high systolic pressure in the systemic arteries. On the other hand, the sudden regurgitation of a portion of the blood from the aorta into the left ventricle leads to a sudden recoil of the blood column and consequently to a low diastolic pressure. The filling of the systemic arteries under great pressure and the sudden drop in this pressure due to the regurgitation can be seen in the short, abrupt pulsation of the visible arteries and can also be felt by palpating the radial artery. The incessant pounding of the hypertrophied left ventricle, the high systolic pressure and the low diastolic pressure tend to produce a very intermittent blood supply in the whole arterial system and especially in the coronary vessels. The latter result is particularly serious as it interferes with the nutrition of the heart muscle and this eventually leads to exhaustion through degenerative changes. When the left ventricle reaches the limit of hypertrophy or becomes exhausted, dilatation ensues and with it relative insufficiency of the mitral valve. This in turn is followed by the usual phenomena of broken compensation.

Symptoms.—Aortic regurgitation may exist in a latent form for years without giving rise to any symptoms whatsoever. Among the earliest symptoms noted are those due to alternations in the circulation, especially cerebral anemia. There may be headaches, attacks of vertigo or flashes of light before the eyes. Anemia is often a marked feature in these cases, the face especially presenting a pale grayish color. Pallor, with a yellow tinge, is suggestive of a syphilitic origin of the lesion. Cyanosis is unusual unless there is an associated mitral lesion. Even prior to any evidence of failing compensation, pain may be a prominent symptom. It may be in the nature of true angina, or a sense of substernal oppression and pain, or it may be an annoying palpitation or throbbing sensation in the precordium. Shortness of breath, especially after exertion, is often the first symptom noticed by the patient. Later, there may be cough and occasionally blood-streaked sputum.

In patients subject to this lesion, distressing dreams and sleep which is broken at frequent intervals, are more common than in any other form of heart disease. Mental symptoms, such as delirium, hallucinations and even suicidal tendencies, are also occasionally encountered.

In cases of aortic insufficiency, sudden death is not infrequent and may occur with but slight premonitory symptoms. The foregoing symptoms are, for the most part, somewhat distinctive of aortic valvular trouble. Failure to maintain compensation may be brought about by a gradual weakening of the left ventricle and the establishing of mitral incompetency. The symptoms then assume the character of those due to broken compensation from any cause, although the symptoms peculiar to the lesion itself may become accentuated.

Physical Signs.—*Inspection.*—A diagnosis of aortic insufficiency can be made in a large number of cases from inspection alone. This is due to the arterial phenomena which constitute the most distinctive feature of the disease.

All of the peripheral arteries are seen to strongly pulsate. Not only is this true of the large superficial vessels but even the small arteries in the hand and face may be seen to throb, and on ophthalmoscopic examination the retinal arteries may be seen pulsating. The arterial pulsation may be so strong that the foot is jerked when the knee is crossed or the head may be moved slightly with each systole.

In addition the so-called capillary pulse is usually present. This is caused by the rapid filling and emptying of the capillaries.

The pulsatile movement may even reach the veins. This may be noted in the veins in the back of the hands. The phenomenon may be brought out by holding the hand so as to drop the wrist. This aids in filling the veins and if pulsation is present it will not be sharp and abrupt as in the arteries, but slow and deliberate. Capillary pulsation is sometimes seen in asthenic conditions accompanied with a low blood-pressure.

In well-marked cases the apex beat is displaced downward and to the left. It is commonly in the sixth interspace but may be as low as the seventh in the anterior axillary line. If the hypertrophy is well-marked, the cardiac impulse is heaving and forcible and may move the entire precordium. In children and young adults the precordium may bulge. If dilatation has occurred the impulse is diffuse and often wavy.

Palpation.—This enables one to appreciate more fully the forcible, heaving character of the impulse. Thrills are not constant but may be felt occasionally. They are either presystolic or systolic at the apex or diastolic and systolic over the base of the heart. The arteries feel large and are usually thickened. When one of the radial arteries is palpated, the pulse beat is felt to impinge against the finger with an abrupt, forcible stroke and immediately recede, hence the terms water-hammer or collapsing pulse. It is also frequently referred to as the Corrigan pulse. The features of this type of pulse are sometimes best appreciated by grasping the wrist and holding it up above the level of the heart, thus accentuating the effect of gravity. The distention of the large vessels at the root of the neck may be so marked as to simulate aneurism. The sphygmographic tracing is characteristic. The upstroke is high and almost perpendicular and forms a very acute angle with the sharply descending downstroke (see Fig. 131). It is in marked contrast to the sphygmographic record of aortic stenosis which is exactly the reverse, namely a slow and low upstroke and a very gradual descent of the downstroke. Auricular fibrillation which is so commonly encountered in mitral disease is not common in aortic lesions unless dilatation of the left ventricle and auricle supervenes.

Percussion.—The area of cardiac dulness is usually increased to the left and in extreme cases may reach the anterior axillary line. The upper border may reach the third rib. There is usually some dulness to the right of the sternum. This may be due to extreme hypertrophy of the left ventricle which causes an extension of the whole heart to the right or it may be brought about by dilatation of the right ventricle when compensation is no longer complete (see Fig. 172).

In some cases a slight area of dulness may be made out, over the sternal end of the second right intercostal space due to dynamic dilatation of the aorta.

Auscultation.—The murmur of aortic insufficiency is caused by the backward rush of blood from the aorta into the left ventricle during the ventricular diastole. While the seat of the production of the murmur is over a point close to the left border of the sternum opposite the third stal cartilage it is, as a rule, not best heard in this situation. The *nctum maximum* for the murmur of aortic incompetency is placed, by ost authorities, at the second costal cartilage on the right, although there are some who believe that the murmur, in many instances, is best heard in the fourth interspace on the left side close to the sternum. Our experience is in accordance with the latter view. The murmur is in most instances soft and low-pitched. It may be very loud, but it rarely has the coarse rasping quality so commonly heard in other valvular murmurs although at times it has a distinct musical tone. The murmur of aortic insufficiency is transmitted downward along the right border of the sternum or towards the cardiac apex. It also may be heard upwards along the sternal margin as far as the sterno-clavicular articulation or in the carotids. In such instances there is usually a dilatation of the aorta associated with stretching of the aortic ring. In well-marked cases, with a loud murmur, it may be heard all over the chest (see Figs. 194 and 196). In some cases the murmur of Duroziez may be heard over one of the large arteries, such as the femoral This murmur is said never to occur except in the presence of aortic leakage. Traube's double sound is sometimes heard in cases complicated by tricuspid insufficiency.

The murmur may be long drawn out and occupy the whole of diastole or it may be short and take up but part of the second sound. Generally speaking, a long loud diastolic murmur shows a considerable degree of pressure kept up in the aorta, which is desirable and a proof that the heart is acting with vigor; also that the valve leakage is not excessive. This is a favorable augury. A weak short murmur indicates the opposite and may be a note of impending danger although there are many exceptions to this (Broadbent).

The pulse pressure may also be utilized to determine the degree of damage. In order to maintain the mean aortic pressure the systolic pressure must be abnormally high in order to compensate for the rapid fall of pressure during diastole. The pulse pressure, however, serves as a useful gauge of the degree of the leak only when compensation is effective.

In some instances the murmur of aortic incompetency is heard by the unaided ear much better than with a stethoscope (Flint).

Associated with the diastolic murmur there is very often heard a *systolic murmur*. This is usually due to some degree of obstruction but it not infrequently happens that a systolic murmur may occur without stenosis. This is explained by Rosenbach as follows: At the beginning of the left ventricular systole the regurgitating flow has not entirely ceased and when this encounters the opposing current a murmur, systolic in time, is produced.

If compensation is complete the first sound at the apex is usually clear. If dilatation of the left ventricle occurs, the first sound at the apex is replaced by a systolic murmur due to relative insufficiency. If compensation is again restored, this murmur disappears and the first sound becomes normal.

In a very considerable percentage of cases of aortic incompetency there is heard at the apex a murmur presystolic in time and which is usually referred to as *Flint's murmur*.

Since Flint's time several hypotheses have been advanced to explain the mechanism of this murmur. (1) That the regurgitant stream tends to lift the great anterior mitral curtain and so to obstruct the mitral orifice at the end of diastole as to impede the current from the auricle. (2) That the mitral valve is thrown into vibration by the two currents, the regurgitant from the aorta and the direct from the auricle. (3) That in the absence of aortic valve disease, but in the presence of adherent pericardium, vibrations may be set up by the current propelled from a dilated and hypertrophied auricle into a ventricle whose muscular walls are deficient in tone. (4) That shortening of the chordæ tendineæ, or dilatation of the left ventricle, may bring about a vertical narrowing of the aperture through which the blood passes from auricle to ventricle, the auricular muscle continuing to be sufficiently powerful to generate a fluid vein.[1] The differentiation of this murmur from that due to mitral stenosis has been considered under the latter condition (see Fig. 136).

Diagnosis.—If due consideration is given to the arterial phenomena associated with the diastolic murmur heard at the base of the heart little or no confusion should arise. If, however, the diagnosis is made on the presence of a murmur alone mistakes may occur as a diastolic murmur is sometimes heard in other conditions. In addition to aortic regurgitation a diastolic murmur may be heard at the base of the heart as the result of insuffiiency of the pulmonary valves. This murmur, sometimes called the *Graham Steell murmur,* is associated with chronic mitral disease and is aused by stretching of the conus arteriosus and the orifice of the pulmonary artery. The time and location of the murmur are all that it has in common with aortic regurgitation. In Graves' disease a diastolic bruit is sometimes heard over the sternum and rarely a cardio-respiratory murmur is diastolic in time. The chief point of distinction between the murmur of aortic insufficiency and other occasional murmurs occurring during the diastolic phase is the presence or absence of the arterial phenomena.

It is not easy to distinguish between aortic regurgitation due to disease of the semilunar valves and relative insufficiency associated with dilatation of the aorta. In the latter condition the murmur is often ransmitted high over the sternum or even into the carotids; in addition here is dulness to the right in the second interspace due to the dilated aorta. The presence of a single or double aortic murmur in a syphilitic hould always call to mind the possibility of a thoracic aneurism. In uch cases an aneurism of the intrapericardial portion of the aorta is not uncommon. The physical signs are not marked and may be entirely absent. Doubtful cases should be examined with the fluoroscope.

The following peripheral *arterio-venous signs* may be met with in cases of aortic insufficiency, and while they are more commonly and strikingly met with in this condition than in any other, they are none of them pathognomonic of this lesion. (1) Collapsing pulse, (2) visible arterial pulsation, (3) capillary pulse, (4) venous pulse, (5) hepatic pulse, (6) femoral snap, (7) double femoral murmur (Duroziez's sign), (8) Double femoral tones (Traube's sign).[2]

AORTIC STENOSIS

Etiology.—This is the rarest of the left-sided valvular lesions. True aortic stenosis unassociated with another valvular lesion is not often ncountered. In the autopsy records of the Pennsylvania and Philadelphia General Hospitals, Norris found 48 instances of aortic stenosis out of a total of 9940 cases having cardiac lesions. It is important to bear this in mind as a systolic murmur at the aortic cartilage is relatively common and on this evidence alone a diagnosis of aortic stenosis is made far too frequently. The interpretation of systolic murmurs heard at the aortic area has been considered on page 788. The great majority of cases of aortic stenosis are encountered in those past the middle period of life

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[1] SANSOM and GIBSON, ALLBUTT and ROLLESTON: "System of Medicine," vol. vi, 362.

[2] TICE, F.: "The Clinical Determination and Significance of Some Peripheral gns of Aortic Insufficiency," *Illinois Med. Jour.*, September, 1911.

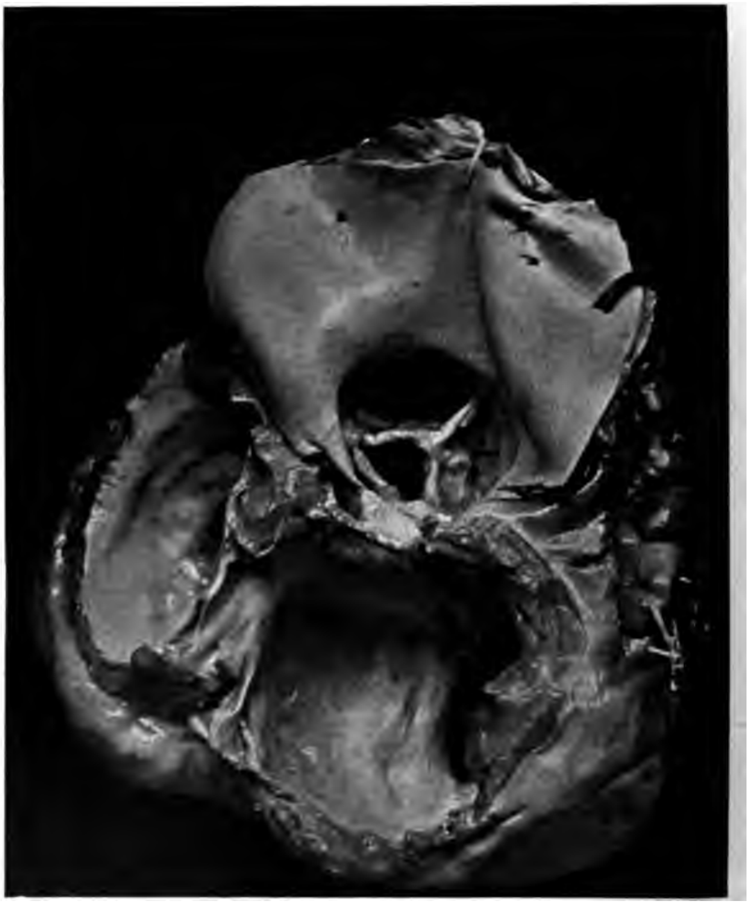
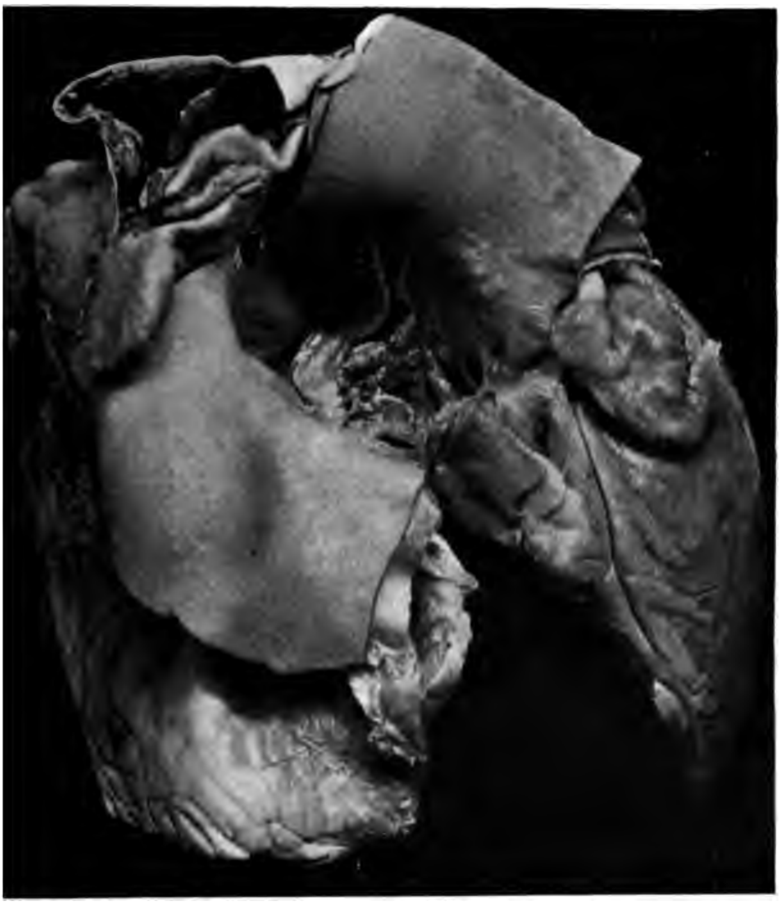


Fig. 404.—Aortic obstruction from above. The margins of the leaflets are fused together, thickened and calcified, reducing the orifice to about one-third of its normal caliber. The mitral and tricuspid orifices are also sclerosed and contracted.

and in those who are the subjects of atheromatous changes in the arteries, especially the aorta. It is rare among women. Occurring as a single lesion it is not often caused by acute rheumatic fever.

Morbid Anatomy.—The anatomical changes which take place in the semilunar valves are precisely similar to those which occur in the intima of the blood-vessels and ending in atheromatous degeneration. As a result, the leaflets become fused, stiff, and rigid and obstruct the free

pe of the blood from the left ventricle into the aorta (see Fig.
. In some instances the obstruction is made worse by vegetations
:h develop during an attack of secondary endocarditis (see Fig.
. The resistance offered adds to the work of the left ventricle which,
consequence, becomes hypertrophied. It is in this variety of Val-
r lesion that the so-called "concentric hypertrophy" is most nearly
·oximated. Practically, however, some degree of dilatation is always



405.—Aortic obstruction, seen from above. The leaflets are fused together, thickened, and indurated. They are covered with small vegetations.

ent. When the heart can no longer overcome the obstruction, dila-
›n and the phenomena of broken compensation ensue.

›athological Physiology.—The mechanism of aortic stenosis is quite
›le. Owing to the obstruction to the ventricular outflow, the
d-pressure in the left ventricle is increased, sometimes to twice
normal. Another effect caused by the obstruction is to prolong
:ime of emptying the ventricle and as a result the ventricular systole

may consume from 5 to 50 per cent. more than the normal time. If the obstruction becomes too great or the muscle becomes exhausted, an insufficient amount of blood reaches the systemic circulation and as a result the pulse pressure becomes small and symptoms of cerebral anemia may develop. In cases of aortic stenosis the left ventricle occupies the same position in maintaining compensation that the right ventricle does in mitral disease. As long as the hypertrophy keeps in advance of the obstruction, the circulatory equilibrium is maintained. If, however, the obstruction becomes too great or, what is more apt to occur, the muscle becomes exhausted, the ventricle dilates and then there develops relative mitral insufficiency with the usual sequence of events which characterize broken compensation. Some observers have expressed the opinion that stretching of the mitral ring and a certain amount of leakage through the auriculo-ventricular opening is to be looked upon as being in the nature of a safety valve action. There is little to support this view, however.

Symptoms.—Of all the valvular lesions aortic stenosis is the most apt to occur in a latent form. It may be present for years without causing serious symptoms. Failure of the left ventricle manifests itself by the occurrence of symptoms which may be brought on by slight exertion, or excitement. They consist of shortness of breath, palpitation, a sense of oppression in the precordium, and substernal pain or anginal attacks. In addition a curtailment of the amount of blood thrown into the systemic arteries may manifest itself by evidences of cerebral anemia, such as vertigo or dizziness. If the left ventricle become dilated from exhaustion, the usual phenomena attending broken compensation are present. Sudden death is not uncommon and may occur as the result of sudden cardiac failure or an attack of angina.

Physical Signs.—*Inspection* does not reveal much of importance; extreme hypertrophy is not often marked as a result of this lesion. Furthermore, occurring as it does in elderly people, the size of the heart is often obscured by a rigid chest wall and emphysematous lungs.

Palpation.—A very common feature of aortic stenosis, although not peculiar to it, is a systolic thrill felt over the base of the heart on the right side. One of the most distinctive features of this lesion is the slow retarded pulse felt at the wrist and graphically shown by a sphygmographic tracing. The pulse is often slow, between 50 and 60 beats per minute and the pulse wave is much prolonged. This is well shown in a pulse tracing but may also be appreciated by the palpating finger as the artery is gradually filled with blood. In addition to the slow slanting upstroke the tracing may show an anacrotic pulse or the pulsus bisferiens.

Percussion.—This is relatively unimportant and is often valueless owing to pulmonary emphysema.

Auscultation.—As we have repeatedly emphasized, a systolic murmur at the aortic area, even when transmitted to the vessels in the neck, is not pathognomonic of true stenosis but is more often associated with changes in the aorta itself, usually a roughening of the intima or a dilatation of the artery. The murmur of true aortic stenosis is transmitted not only upward to the carotids but is frequently audible along the right border of the sternum as low as the fourth or fifth ribs. In some instances it is very loud and may be heard all over the precordium and in such instances the question always arises as to whether the murmur heard at the apex is the same as that heard at the aortic cartilage or is a second murmur due to mitral insufficiency. Even more distinctive of stenosis than the slow retarded pulse is the character of the second aortic sound. This is either very feeble or inaudible due to the fact that the stiff and rigid valve segments are incapable of snapping together. If, therefore, the second aortic sound is normal and especially if it is loud and ringing in character, the murmur originates in the aorta and is not due to rigid and diseased valves. Some leakage is probably always present but this is often so slight that no murmur can be heard.

Diagnosis.—The diagnosis of true aortic stenosis rests upon the following facts: (1) A systolic thrill felt over the base of the heart to the right of the sternum; (2) a slow retarded pulse; (3) a systolic murmur heard at the aortic cartilage and transmitted to the vessels of the neck; and last, but most important, a feeble or inaudible second aortic sound.

TRICUSPID INSUFFICIENCY

Etiology.—Lesions involving the tricuspid valve may be (1) organic and (2) functional.

1. *Organic disease* of the tricuspid leaflets is rare although not so unusual as was once believed to be the case. Formerly it was taught that organic lesions of the valves in the right heart were almost invariably congenital in origin. Recent investigations, however, have shown that these lesions are more frequently the result of acquired disease than of a congenital defect.

(*a*) The tricuspid leaflets may be the seat of an acute inflammatory process which develops during the course of one of the acute infections such as puerperal fever and other forms of general sepsis, gonorrhea, pneumonia, diphtheria, scarlet fever and acute rheumatic fever. The last-named infection is by far the most important etiological factor. Statistical studies indicate that acute rheumatic fever is the apparent cause of organic tricuspid disease in from 35 to 62 per cent. of cases.[1] Occasionally the acute endocarditis is limited to the tricuspid leaflets but in the majority of cases it is associated with similar lesions of the aortic or mitral valves, especially the latter; with few exceptions the valves in the left heart are the sites of old lesions.

(*b*) Thickening of the tricuspid leaflets as the result of fibrosis occurs under practically the same circumstances as similar lesions affecting the mitral valve.

(*c*) The organic lesion may be the result of rupture of one of the leaflets or chordæ tendineæ following a blow on the chest or unusual muscular effort. This is extremely rare.

2. *Functional or Relative Tricuspid Insufficiency.*—This is extremely common and may arise in a large number of conditions. The functional efficiency of the tricuspid valve depends very largely upon the tone of the sphincter-like muscular fibers surrounding the orifice. It has long been recognized that from a mechanical standpoint the tricuspid valve is much inferior to the mitral. Even a slight degree of dilatation of the ht ventricle induces incompetency. This is due not only to relaxation

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[1] GRIFFITH: *Edinburgh Med. Jour.*, 1903, lv, 105. HERRICK: *Boston Med. and Surg. Jour.*, 1897, cxxxvi, 245. LEUDET: Paris Thesis, 1881. NORRIS: "Studies in Cardiac Pathology," 1911. PITT: "Allbutt's System of Medicine," 2d ed.

of the tricuspid sphincter, but more especially to the fact that in dilated hearts the origin of the chordæ tendineæ and the papillary muscles are too far from the center of the ventricular cavity to permit perfect valvular closure (Krehl). Indeed Mackenzie inclines to the opinion that the tricuspid valve is barely able to close the orifice perfectly and that the slightest disturbance will cause some leakage. Thus the transient tachycardia which sometimes results from the excitement incident to a physical examination may be accompanied by a tricuspid systolic murmur which is usually very fugitive and disappears when the heart becomes quieter. The ease with which regurgitation occurs through the right auriculoventricular orifice is considered by some to be in the nature of a safety valve, whereby the thin wall of the right ventricle is relieved of undue tension. Experimentally it is not possible to raise the tension in the right ventricle on account of the ease with which leakage occurs through the tricuspid orifice (see Figs. 205 and 206).

The essential factor in the production of functional tricuspid insufficiency, therefore, is an increase in tension which in turn tends to dilate the right heart. This may be brought about by work which necessitates great muscular and intrathoracic straining, asphyxia, obstruction in the pulmonary circulation, such as may occur in chronic inflammatory affections of the lungs and, lastly, and most important, by disease of the heart itself. Dilatation of the right ventricle and functional tricuspid insufficiency inevitably occur whenever failing compensation arises as the result of chronic valvular or myocardial disease. Disease of the mitral valve is especially apt to bring about these conditions. Slight degrees of tricuspid insufficiency are also of frequent occurrence in all diseases in which the nutrition of the cardiac muscle suffers. Among such diseases may be mentioned the acute specific fevers, diabetes, the cachexia accompanying malignant disease, and severe anemia. It is probable that some degree of tricuspid insufficiency occurs in every dying or failing heart.

Morbid Anatomy.—When the insufficiency is due to organic disease of the valves, the lesions are essentially the same as occur in the mitral valve. The leaflets may be deformed or may be the seat of fibroid changes, vegetations or ulcerations. The changes occur oftenest on the free margin of the middle portion, with the anterior portion next in frequency. The chordæ tendineæ are often attenuated and elongated and the tip of the papillary muscle not infrequently is indurated. The tricuspid opening is enlarged.

Whether the incompetency is due to organic disease or functional incapacity of long standing the right auricle is much dilated and globular in appearance and the right ventricle is also much dilated. The ventricle may show also a certain amount of muscular hypertrophy. Inasmuch as the lesion produces a damming back of blood in the venous system the viscera (liver, spleen and kidneys) show evidences of chronic congestion. If the congestion of the liver is constant, degeneration and atrophy of the liver cells as the result of pressure often occurs, producing a condition known as the "nutmeg" liver.

Pathological Physiology.—The first effect of tricuspid regurgitation is to distend the right auricle. As the wall of this chamber is very thin and contains but little muscular structure, dilatation quickly ensues if the back flow is prolonged. As the right auricle is unable to withstand the pressure the blood is dammed back into the venæ cavæ giving rise to the characteristic features of tricuspid insufficiency. The venous stasis which results may give rise of edema of the extremities and effusions into the serous cavities (see "Hydrothorax," p. 614 also Figs. 109, 206); to congestion of the kidneys causing diminished excretion of urine which contains large quantities of albumen and casts; to congestion of the liver which becomes enlarged, painful and often pulsatile; and to congestion of the face and pulsation of the jugulars. In addition an asphyxial condition may be produced by stasis in the medulla oblongata.

Stadler[1] has shown experimentally in rabbits that tricuspid insufficiency, if marked, leads to dilatation and hypertrophy of both the right auricle and ventricle and to atrophy of the left ventricle, due apparently to the smaller amount of blood handled. In the less severe lesions the tricuspid insufficiency is apparently compensated for by increased vascular tone and hypertrophy of the right auricle, which not only contracts more forcibly but aspirates blood from the veins more powerfully.

Symptoms.—When the tricuspid insufficiency is transient, such as occurs as the result of slight excitement, there are no symptoms. In well-defined cases the most constant symptom is dyspnea. This may become manifest only after exertion but in extreme cases it is apparent even when the patient is at rest. In severe cases the patient may be orthopneic and often is drowsy. The slightest exertion causes fatigue. Precordial pain is not often complained of but there may be a sense of oppression in the chest. Pain over the liver is a common and important symptom. It is caused by engorgement of the liver and stretching of the capsule. If the engorgement is of long standing, the patient may be slightly jaundiced. Associated with the hepatic pain there is often anorexia, nausea and vomiting and eructations. It not infrequently happens that the prominence of the gastric symptoms deceives a careless observer into the belief that the stomach is the real seat of the patient's symptoms.

Edema of the lower extremities is common. The condition is less evident after a night's rest in bed. Ascites and effusions into the serous cavities of the thorax may occur alone or in association with the edema. The urine is scanty and contains both albumen and casts. The chest should always be carefully examined in patients suffering from tricuspid regurgitation as effusions into the pleural sacs, especially the right, are common and may be responsible for much of the dyspnea.

Physical Signs.—*Inspection.*—In a patient with well-marked tricuspid insufficiency *cyanosis* is a marked feature. The face has a livid hue while the lips, tips of the ears and finger ends are bluish in color. If the venous stasis is of long standing the conjunctiva may have an icteroid tinge or the skin may be slightly jaundiced. The latter symptom is always of ominous import. The veins of the neck, arms and chest are often dilated. In some cases a distinct systolic pulsation of the liver is visible. Edema of the extremities and genitalia is commonly present (see Figs. 208, 399).

The *cardiac impulse* is often diffuse and the apex beat is seen or felt beyond the mid-clavicular line. Marked cardiac pulsation due to the hypertrophied right ventricle may be seen on the right side between the sternal and parasternal line.

Pulsation in the jugular veins is an important diagnostic sign. Even without tracings three pulsations may be seen in the supraclavicular triangle. (1) A wave appearing slightly before the impulse at the apex beat, due to pulsation of the right auricle; (2) a wave which is synchronous with the beat in the carotid artery, as felt higher up in the neck; (3) a wave occurring immediately after systole of the ventricle, the ventricular wave (Osler and Gibson). The ventricular wave is produced as follows: When the tricuspid insufficiency has become marked the regurgitation of blood becomes too great for the auricle to contain. The latter dilates to such an extent that the orifices of the veins emptying into it cannot be closed. As a result there is during each systole of the right ventricle an unobstructed path from the ventricle to the veins. Such being the case, with each contraction of the right ventricle the impulse of the blood regurgitating through the tricuspid opening is imparted to the blood in the jugulars. In like manner the systolic pulsation of the liver is also produced. When the leakage becomes very marked, the auricle becomes paralyzed and the auricular fibrillation is present. The transition of the normal venous pulse to that characterized by the ventricular pulse is shown diagrammatically in Fig. 406.

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[1] *Deut. Arch. f. Klin. Med.*, 1905, lxxxiii, 1 and 2.

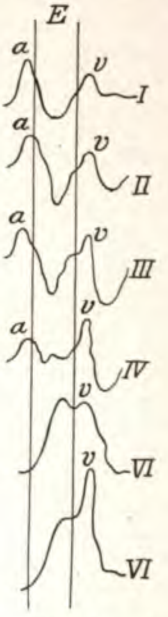

FIG. 406.—Diagram to show the transition from the normal venous pulse to the ventricular form, *i. e.*, in tricuspid insufficiency. *I* is the normal venous pulse from which the c wave, occurring between *a* and *v*, probably due to the carotid artery, has been omitted; *a*, auricular wave; *v*. ventricular wave, whose significance in the normal venous pulse is doubtful. *E*, period of outflow into the pulmonary artery. (*After Mackenzie.*)

Palpation.—Marked pulsation may be felt over the epigastrium and to the right of the sternum in the fourth and fifth interspaces. A systolic thrill is often felt and occasionally a slight systolic shock may be perceived. Pulsation may be felt over the liver. When the liver is enlarged the surface is smooth and the edge firm and sharp. The radial pulse is usually small, weak and often irregular. The blood-pressure is normal or slightly below normal.

Percussion.—The area of cardiac dulness is increased in the transverse diameter, especially to the right of the sternum. In extreme cases the dulness may extend three fingers' breadth or more, to the right of the sternum. Relative tricuspid insufficiency is believed by some to produce a wider area of dulness than is the case if an organic lesion is present.

Auscultation.—The punctum maximum of a tricuspid murmur due to insufficiency is over the middle of the lower part of the sternum. It is systolic in time and may be best heard a little to the left or a little to the right of the sternum; and if hypertrophy of the right ventricle is marked it may be audible all over the precordium. In those cases of relative insufficiency of brief duration the murmur is the only evidence of tricuspid leakage we possess. On the other hand, it is to be borne in mind that an extreme degree of regurgitation may be present without a murmur being heard. A weak muscle and a widely dilated orifice may not produce a bruit.

If due to organic disease, the murmur may be rough in character. When due to relative insufficiency, the murmur is usually soft and blowing in character. The tricuspid murmur is not often transmitted far from the area of maximum intensity. Occasionally it is heard as far as the pulmonary area. It may be transmitted to the right and at times is audible posteriorly near the angle of the right scapula. The murmur may replace all or part of the first sound. The character of the sounds at the base of heart vary considerably. They may be feeble or accentuated.

Diagnosis.—The diagnosis of tricuspid insufficiency depends on the presence of increased cardiac dulness to the right of the sternum; a systolic murmur best heard at the xiphoid cartilage; a venous pulse of the ventricular type; pulmonary congestion and enlargement of the liver with systolic pulsation.

If the regurgitation is functional in type and due to trivial causes there may be nothing to indicate the condition except a soft, blowing murmur, transient in character. If, on the other hand, the functional derangement is severe, there may be no murmur as the widely dilated orifice and weak muscle do not permit of sufficient driving force to cause a murmur. In such cases the diagnosis depends on the presence of the ventricular type of pulse, enlarged right heart and engorged liver. If the murmur is organic in origin, it will be rough in character and as compensation is restored will persist instead of disappearing as is the case with murmurs due to relative insufficiency.

If the tricuspid murmur exists alone, which is rarely the case, there is little difficulty in determining its origin. As a rule, however, it is combined with other murmurs and in such cases the punctum maximum of each murmur must be determined.

TRICUSPID STENOSIS

Etiology.—Stenosis of the tricuspid orifice is relatively rare and for the most part is first recognized at the autopsy table. The lesion rarely exists alone. W. W. Herrick[1] has collected 187 cases and of this number the tricuspid valve was involved alone in 14. In 102 there was disease of the mitral valve as well and in 64 the aortic valve was also involved.

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[1] *Arch. Int. Med.*, 1908, ii, 295.

At the Philadelphia General Hospital among 8640 autopsies Norris found 8 cases of tricuspid stenosis recorded.

The most important etiological factors are acute rheumatic fever and chorea. In Herrick's series 61 cases gave a definite history of acute rheumatic fever and in 11 more there was a doubtful history of this infection or chorea. Pitt found the incidence of acute rheumatic fever to be as high as 62.06 per cent. Syphilis has been believed to be the exciting cause in a few cases. Rarely a tumor or a fibrinous ball may obstruct the tricuspid orifice.

Hirschfelder[1] believes that when the tricuspid stenosis is associated with mitral stenosis the same etiological factors are concerned. "In view of the work of Goodhart, Roy and Adami, and Weber and Degny, it is not unlikely that the overstrain of the right ventricle, brought about by the latter conditions, leads to edema and hemorrhage into the tricuspid valve, and that these processes usher in fibrosis. In other words, the mitral stenosis itself becomes an etiological factor in the tricuspid lesion, and the pathological process completed in the mitral is now transferred back one step in the circulation and repeats itself in the tricuspid."

Pitt[2] gives the following age distribution in 109 cases:

Under 10 years	1 case
Between 11 and 20 years	19 cases
Between 21 and 30 years	41 cases
Between 31 and 40 years	29 cases
Between 41 and 50 years	12 cases
Between 51 and 60 years	4 cases
Between 61 and 70 years	3 cases
	109 cases

As but one of the 109 cases was under ten years of age, the belief once currently held that such lesions are commonly congenital, does not receive much support.

Tricuspid stenosis is much more frequent in women than men. In Herrick's series there were 133 women and 38 men.

Morbid Anatomy.—As to the valves themselves the structural changes are analogous to those found in the mitral valve when stenosis is present. There may be vegetations or sclerosis, and changes in the chordæ tendineæ or papillary muscles. Obstruction of the tricuspid orifice is similar to that occurring in the mitral except that such high degrees of contraction are rarely found; nearly always the opening will admit two fingers (see Figs. 407 and 408).

The condition of the heart will depend on the duration of the obstruction. During the first stage there will be some hypertrophy of the auricular wall and of the superior vena cava. At a later stage the caval opening is found to be greatly enlarged, so that the auricle and the veins form a continuous sac. The auricular wall then atrophies and may become a mere fibrous sac formed by the endocardium and epicardium. Pericarditis, sometimes acute, but more frequently of the chronic adhesive type, is a very common finding.

Owing to the fact that mitral stenosis is almost constantly associated with tricuspid stenosis (85 out of 87 cases analyzed by Pitt) it is often difficult to determine how much or how little effect the mitral lesion has had in bringing about the secondary changes in the heart.

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[1] "Diseases of the Heart and Aorta," 2d ed.

[2] ALLBUTT and ROLLESTON: "System of Medicine," vol. vi, p. 331.

The liver usually presents the appearance of chronic stasis but is not always enlarged. The liver may in fact be smaller than normal as the esult of cirrhosis and perihepatitis due to the prolonged stasis.

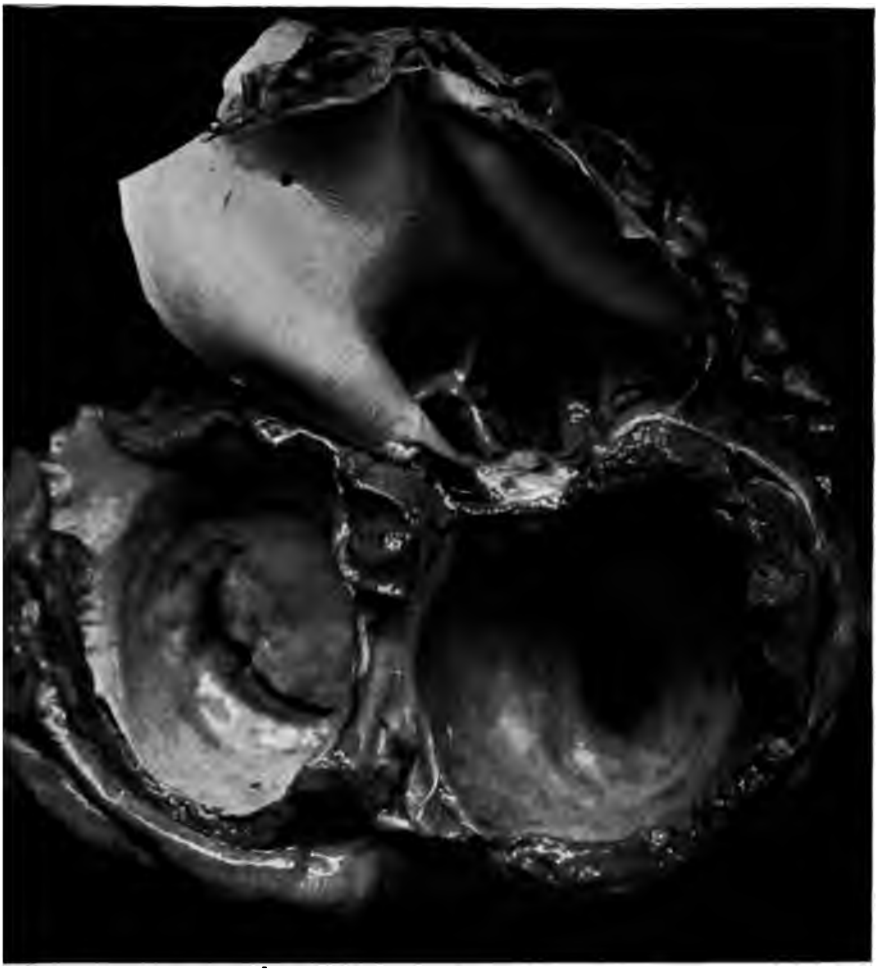

Fig. 407.—Stenosis of the aortic, mitral, and tricuspid valves. The aortic leaflets re fused together and the orifice is greatly reduced in caliber. The mitral orifice consists f a mere slit, of crescentic outline—buttonhole type. The tricuspid orifice is also conracted. (*Specimen from the Philadelphia Hospital.*)

Pathological Physiology.—With the onset of the stenosis the wall of he right auricle hypertrophies as the result of the increased amount of work it has to perform. Owing to the increased strength of the right uricle the auricular or presystolic wave is large and well marked. Macenzie refers to a case in which the auricle had become so greatly hyperrophied that it sent back a large wave into the jugular vein, and with such force that he was able to hear the valves in the jugular and subclavian veins close with a snap. Hypertrophy of the right auricle also sends a wave back into the veins with such force that distention of the liver and pulsation of that organ is brought about. Later when the auricle becomes overdistended and paralyzed, the veins remain per-

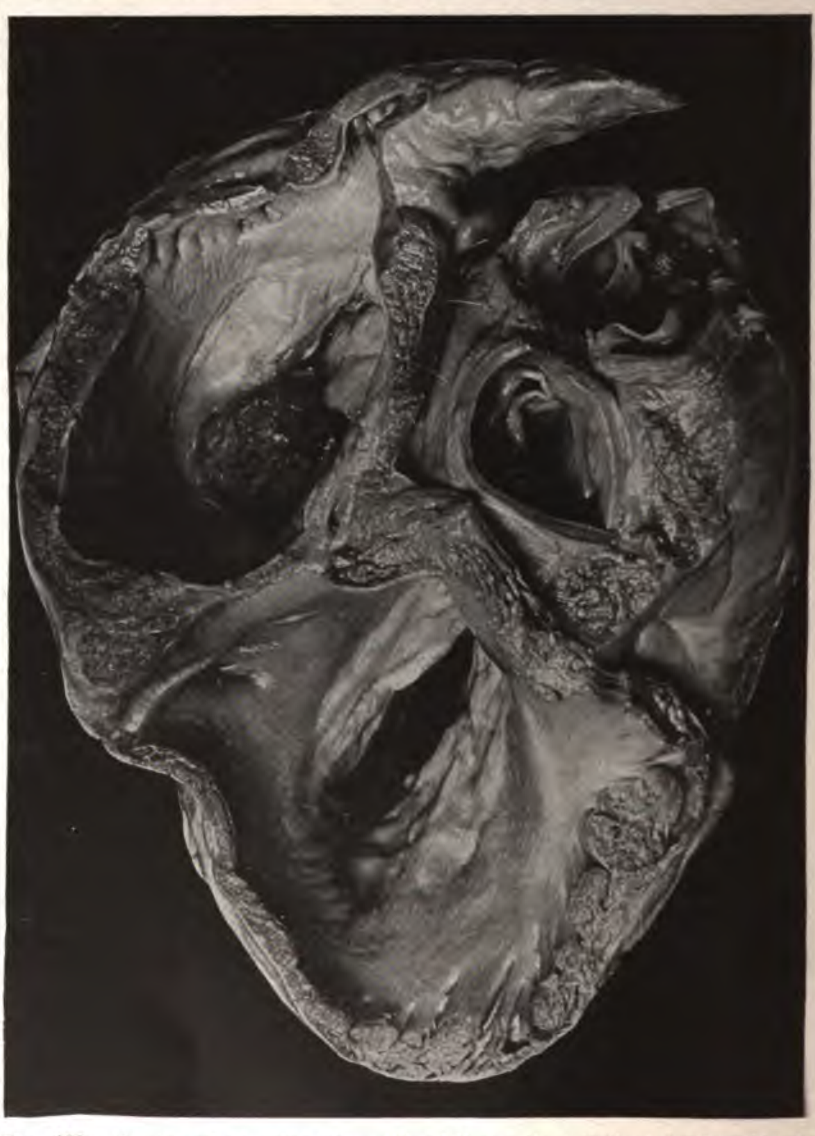

Fig. 408.—Acute and chronic endocarditis of the aortic, mitral, and tricuspid valves. The upper portion of the auricles has been cut away. The three valvular orifices thus exposed show induration and contraction of their component tissues. The mitral valve in addition shows a large dark mass of vegetations of recent origin which almost completely occlude its orifice. The endocardium and myocardium of both auricles are thickened. (*Photograph by Dr. Alfred R. Allen.*)

nanently full and cease to pulsate. Damming back of the blood in the eins increases the pressure in these vessels; interferes with the circulaion of the blood through the heart and lungs; produces cyanosis; and inally may cause polycythemia.

The slightest overstrain may aggravate the venous stasis and bring bout extreme dyspnea, cyanosis, edema and effusions into the serous avities. These symptoms may pass off quickly when the patient is ut at rest.

Symptoms.—Pitt gives the symptoms of tricuspid stenosis in the rder of their relative frequency as follows: dyspnea, edema, albuminuia, enlarged and tender liver, and cyanosis. Dyspnea is almost inariably present; edema and albuminuria in two-thirds of the cases; and nlarged liver and extreme cyanosis in one-half of the cases. Pain raditing down the arms or over the hepatic region is quite common. Hemptysis is not infrequent but is to be looked upon as being due to the ssociated mitral lesion.

The lesion may exist for years without causing symptoms or at the nost, slight shortness of breath on exertion.

Physical Signs.—*Inspection.*—More or less cyanosis is present; in ome cases it is very marked. In addition to the cyanosis the patient ften has a congested appearance due to *polycythemia*. The red count nay be as high as 8,000,000 or 9,000,000 per cubic millimeter. Clubbing f the fingers is often a marked feature. The veins in the lower part of he neck are usually dilated and the jugular bulb may stand out promiently. It will be recalled that the venous pulse in tricuspid regurgitaion is ventricular in origin and systolic in time; that in tricuspid stenois is auricular in origin and presystolic in time. Mackenzie considers ulsation of the liver with a marked wave due to the auricle as an evilence of possible tricuspid stenosis. When the auricle becomes overdisended and paralyzed the pulsation disappears.

As tricuspid stenosis is almost invariably associated with stenosis of the mitral valve, it is difficult to determine which lesion is most responsible for the diffuse area of pulsation and displacement of the apex beat to the left.

Palpation.—In some instances a thrill, presystolic or systolic is felt over the lower part of the sternum.

Percussion.—The transverse diameter of cardiac dulness is always increased, especially to the right of the sternum.

Auscultation.—It is only rarely that a presystolic tricuspid murmur s heard and for this reason the diagnosis is not often made during life. n Leudet's[1] series of 114 cases the diagnosis was made before death in ut 6 instances. The murmur when present is best heard at or to the ft of the xiphoid cartilage; is presystolic in time; and is not transmitted. lthough rough in character, it is less harsh than the mitral presystolic urmur. Pitt states that a presystolic murmur has been noted in less an 10 per cent. of the cases and that a localized systolic murmur has een much more frequently noted. In half the cases there is no murmur t the tricuspid area.

Diagnosis.—This cannot be made with any certainty. The condition to be suspected if there be marked cyanosis, clubbing of the fingers, yspnea, edema of the extremities, auricular pulsation of the jugulars and liver and the presence of a presystolic murmur over the lower part of the sternum.

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[1] Paris Thesis, 1881.

PULMONARY INSUFFICIENCY

Etiology.—This is a rare lesion. Norris found but one case among 8640 autopsies at the Philadelphia General Hospital and Hirschfelder records 3 cases among 24,000 medical admissions to the Johns Hopkins Hospital. Pitt, in 1910, placed the total number of cases on record as 109.

The lesion affects both sexes equally, may occur at any age, but in the great majority of cases it occurs in adult life.

Pulmonary insufficiency may occur: (1) As the result of an acute endocarditis. The infecting organism is usually the streptococcus, pneumococcus or the gonococcus. Pitt has called attention to the relative frequency of *gonorrheal endocarditis* affecting the pulmonary valve. (2) In association with aneurism of the aorta in which the inflammatory change in the neighborhood of the pulmonary artery causes an adhesion of one or more cusps of the pulmonary valve to it (Pitt). (3) As the result of fibroid changes in the valve leaflets. The fibrosis may produce insufficiency alone or it may be associated with stenosis. (4) As the result of congenital malformation or rupture of a leaflet. (5) Relative insufficiency may occur if pressure in the pulmonary artery is much increased. This may arise in association with any condition which produces long-continued pulmonary hypertension, such as emphysema, chronic pulmonary disease and especially mitral stenosis. Graham Steell has emphasized the frequency with which dilatation occurs in association with mitral stenosis.

Pathological Physiology.—Incompetence of the pulmonary valve leads to the regurgitation of blood into the right ventricle under the circumstances that insufficiency of the aortic valve allows blood to flow back into the left ventricle. As a result of this leakage the right ventricle becomes hypertrophied. If, however, the leakage becomes too great, dilatation occurs and there is added to the clinical picture, tricuspid insufficiency.

Morbid Anatomy.—When the pulmonary valve is incompetent, the structural changes are usually less well marked than those encountered in the left heart. The leaflets are less thickened and the sclerosis of the endocardium is less markedly evident. Congenital abnormalities, principally in the form of an increase or diminution of the leaflets, may be the cause of the insufficiency. As a rule, these abnormalities are unproductive of leakage.

The right ventricle is generally dilated as is also the conus arteriosus. The viscera show changes similar to those occurring from failing compensation in other cardiac lesions.

Functional insufficiency due to dilatation of the pulmonary artery and valve orifice may be transient or permanent. For this reason pulmonary insufficiency due to stretching of the pulmonary orifice may not be demonstrable until post-mortem unless the condition is permanent. Permanent stretching of the pulmonary artery is encountered most frequently in association with mitral stenosis. Testing the efficiency of the pulmonary valve by pouring water into the artery in the excised heart and measuring the diameter of the pulmonary orifice are of doubtful value.

Symptoms.—As long as the right ventricle compensates for the akage there are not apt to be any symptoms. If other valvular lesions e present, the symptoms may be attributed to them and the pulmonary ouble overlooked entirely. Dyspnea, especially on exertion, is perhaps e most common symptom and this may be paroxysmal in character. wing to the disturbance of the pulmonary circulation, there is often ugh. If the lesion is due to acute endocarditis, emboli frequently are dged in the lungs. In such cases hemoptysis is common.

In cases of general sepsis the pulmonary valve may be involved alone in association with valvular lesions elsewhere. All of the valvular eas should be carefully examined in the presence of pyemic symptoms.

Physical Signs.—*Inspection.*—Cyanosis may be present. The apex at of the heart is displaced downward and to the left. Pulsation is arked in the epigastrium due to the hypertrophied right ventricle and the second and third interspaces due to dilatation of the conus arteosus. Jugular pulsation may be noted also.

Palpation.—Marked pulsation is felt in the second and third interaces to the left of the sternum, due to the beating of the conus arterious. Rarely a diastolic thrill may be felt.

Percussion.—The transverse diameter of cardiac dulness is noticebly increased.

Auscultation.—The murmur of pulmonary insufficiency is diastolic n time and usually best heard at the second costal cartilage on the left. he punctum maximum, however, may be midway between the nipple nd the sternum (Bryant). The murmur may be soft and of brief uration or it may be loud, rough and replace the second sound. It ounds more superficial than the diastolic murmur due to aortic incometence. The murmur of pulmonary insufficiency is localized or slightly ransmitted down the left border of the sternum. It is not transmitted nto the vessels of the neck; is intensified by the erect posture; and, according to Gerhardt, is intensified by expiration. In some cases the vesicular nurmur may be jerky in character.

Diagnosis.—Although there are a number of physical signs which re said to be more or less distinctive of pulmonary insufficiency, the ecognition of the condition is fraught with difficulty. It is to be disinguished from aortic insufficiency by the following points: A diffuse ather than heaving, forcible cardiac impulse; absence of the capillary nd water-hammer pulses and large arterial pulse pressure; location f murmur to the left of the sternum; absence of transmission into the essels of the neck; and an increase in the intensity of the murmur during xpiration.

In cases of mitral stenosis with a *diastolic murmur* to the left of the ternum it should be borne in mind that the murmur may be due to lilatation of the pulmonary artery rather than aortic regurgitation. abot[1] has emphasized the frequency with which a diastolic murmur o the left of the sternum occurs in association with mitral stenosis. Ie was unable, however, to demonstrate at autopsy that it was due to ilatation of the pulmonary artery. Its differentiation from the murmur f aortic regurgitation will depend on the presence or absence of the rterial phenomena characteristic of the latter condition (p. 732).

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[1] *Trans. Assoc. Am. Phys.*, 1914.

PULMONARY STENOSIS

As an acquired lesion pulmonary stenosis is exceedingly rare, but as the result of a congenital defect is relatively common. The latter will be considered under congenital lesions of the heart.

Etiology.—Acquired disease of the pulmonary valves may be the result of acute rheumatic fever or some other of the acute infectious diseases or sclerotic changes which lead to thickening of the valve leaflets and later to atheromatous degeneration. Pulmonary stenosis, whether acquired or congenital, seems to predispose to *pulmonary tuberculosis.* Among 449 cases of pulmonary stenosis analyzed by Norris, tuberculosis of the lungs was noted in 160.

Morbid Anatomy.—The changes are analogous to those seen in the aortic valve. If the disease is acute, vegetations may be present and in some instances are so large as to nearly block the orifice. In other instances the inflammatory process is evidently chronic in nature and structural defects result from induration, thickening, fusion, or calcification of the valve leaflets. The conus of the right ventricle is involved in a large proportion of cases. In addition to the valvular defect, hypertrophy and dilatation of the right auricle and ventricle are usually present. With the onset of failing compensation tricuspid insufficiency occurs and with it the usual phenomena which attend failing compensation in other cardiac lesions.

Pathological Physiology.—The partially obstructed pulmonary orifice increases the work of the right ventricle which becomes hypertrophied. If the stenosis is marked, the volume of blood forced into the pulmonary circulation is diminished. This leads to venous stasis and the marked cyanosis which is characteristic of the condition. When the right ventricle fails to compensate for the defect, dilatation takes place and with it tricuspid insufficiency.

Symptoms.—The acquired form does not give rise to very marked symptoms so long as the compensation is maintained. Varying degrees of shortness of breath may be present on exertion. Slight cyanosis and edema may be present but are usually not noted until tricuspid insufficiency occurs and compensation fails.

Physical Signs.—*Inspection.*—Unless failure of compensation has occurred, nothing is to be noted on inspection. With the onset of failing compensation the evidences of venous engorgement which follow tricuspid insufficiency will be seen. Clubbing of the fingers is not often present in the acquired form.

Palpation.—A systolic thrill may be felt in the second and third interspaces to the left of the sternum.

Percussion.—Cardiac dulness is increased to the right of the sternum as the result of hypertrophy or dilatation or both, of the right ventricle.

Auscultation.—The characteristic feature is the presence of a murmur systolic in time and best heard at the pulmonary area. It is usually transmitted upward and to the left toward the clavicle. The murmur is usually harsh in character and gives the impression of being superficially placed. The second pulmonic sound is either very feeble or entirely absent. If pulmonary insufficiency is also present, the second pulmonic sound may be replaced by a diastolic murmur.

Diagnosis.—Acquired pulmonary stenosis is so exceedingly rare that a diagnosis of the condition is permissible only when every means has been taken to determine the source of the murmur. It is to be borne in mind that systolic murmurs heard at the pulmonary area are extremely common. In the great majority of instances they are functional in character but occasionally are due to organic lesions situated elsewhere.

Not infrequently the murmur of aortic stenosis is best heard to the left of the sternum. In such cases the pulmonic second sound is either normal or accentuated instead of being absent or very faint. The murmur of aortic stenosis is transmitted to the vessels of the neck while that of pulmonary stenosis is not.

Oille, Graham and Detweiler[1] have called attention to the fact that systolic murmurs at the pulmonary area are not infrequently due to mitral insufficiency. In such cases there is no murmur at the apex. Even when a systolic murmur due to mitral insufficiency is heard at the apex it is often loudest at the pulmonary area over the left auricular appendage (see Fig. 163). Rarely a loud systolic murmur due to a patent ductus arteriosus is heard to the left of the sternum. This will be referred to under congenital heart lesions.

Functional murmurs, systolic in time and best heard at the pulmonary area may be present in any condition associated with anemia. Often a transient systolic murmur is heard when the heart is overacting as the result of nervousness or exertion.

Just as in the case of aortic stenosis, the character of the second sound is the important feature. If true stenosis is present the second sound is feeble or absent; in all other instances, it is normal or accentuated.

[1] *Jour. Am. Med. Assoc.*, Oct. 2, 1915.

CHAPTER XXVIII

CONGENITAL HEART DISEASE

"Congenital cardiac disease may be defined as that condition in which, through arrest of development or disease occurring in intrauterine life, anomalies in the anatomical structure of the heart or great vessels exist, leading to irregularities in the circulation. It is frequently associated with congenital cyanosis and clubbing of the fingers, and constitutes in extreme cases the *morbus cæruleus* of the older writers" (Maude E. Abbott).

For the most part congenital lesions of the heart are of pathological interest only. In some instances the defect is so great as to be incompatible with life; in others, the individual may reach adult life without there being any evidence of cardiac trouble, or there may be distinctive symptoms; in still others, the defect may consist of some abnormality from which there is no disturbance of function whatever, as for instance the presence of four or of two leaflets in the aortic or pulmonary valves. Supernumerary leaflets occur more frequently in the pulmonary valve than the aortic while the presence of two instead of three leaflets occurs more often in the latter (19 out of 21 cases observed by Osler).

The chief clinical interest in congenital defects, which are not incompatible with life, is that they seem extremely prone to become the seat of an acute endocarditis or, as in the case of anomalous valves, to become the seat of sclerotic changes.

The best presentations of the subject are those of Maude E. Abbott[1] and Lawrence Humphry.[2] Only the more common defects will be considered here.

Symptoms of Congenital Heart Disease.—Symptoms may be present from birth or they may not appear until the child is a year or more old. The first symptom to be noted is cyanosis which is present in about 90 per cent. of the cases. This symptom is so characteristic of congenital disease of the heart that the terms "blue disease" and "morbus cæruleus" are synonymous. The cyanosis is most noticeable in the face, hands and feet which are of a livid or bluish tint. Usually the cyanosis is most marked in the nose, ears, lips and the tips of the fingers and toes. The mucous membranes may present a cyanotic tinge also. The cause of the cyanosis is not altogether clear and various hypotheses have been advanced. It has been suggested that the condition is caused by: (1) The mixture of venous and arterial blood; (2) to deficient aeration of the blood; and (3) general venous congestion. The latter view is the one most generally held, although it cannot be said to cover all cases. There are not a few instances in which no satisfactory explanation is forthcoming. Associated with the cyanosis there is often a marked *polycythemia.* The red cells may be as high as 11,000,000 per cubic millimeter. In addition to the high red cell count the percentage of hemoglobin is increased and the specific gravity of the blood is abnormally high. Hemorrhages sometimes occur.

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[1] OSLER'S "Modern Medicine," vol. iv, 1st ed.

[2] ALLBUTT and ROLLESTON: "System of Medicine," vol. vi.

Clubbing of the fingers, usually of an extreme degree, is a very frequent manifestation. Clubbing of the fingers and cyanosis in a small child are of themselves sufficient evidence upon which to base a diagnosis of congenital heart disease (see Fig. 1). Dyspnea may be a marked feature; it may occur only as the result of overexertion or it may be paroxysmal in character. Cough may be present and is apt to be brought on or increased by exertion.

The surface temperature is usually low and this is especially noticeable in the hands and feet. In the majority of cases of congenital disease the child is weakly and succumbs early in life. In those who reach adult life, stenosis of the pulmonary orifice is the lesion most frequently present.

In many cases the physical signs are marked, especially the presence of a murmur. The existence of the latter in association with one or more of the characteristic symptoms renders the diagnosis of congenital disease easy, although it may be difficult and often impossible to determine the exact nature of the defect.

Congenital Pulmonary Stenosis and Atresia of the Pulmonary Artery.—Stenosis of the pulmonary orifice acquired in adult life is exceedingly rare. Congenital stenosis, on the other hand, is relatively common. It may be the result of fetal endocarditis or of anomalies in the development of the heart. Of 366 cases of congenital lesions reported by Peacock and Keith 254, or 69 per cent., were due to pulmonary stenosis.

The obstruction may be due: (1) To constriction of the orifice, with or without atresia or obliteration of the artery; (2) to obliteration or narrowing of the pulmonary artery: and (3) to stenosis of the conus arteriosus. In the majority of cases the stenosis is associated with other developmental anomalies the most frequent of which are patency of the foramen ovale and ductus arteriosus, an opening in the ventricular septum and transposition or malposition of the aorta and pulmonary artery.

Symptoms and Physical Signs.—The two distinctive features are cyanosis and clubbing of the fingers and toes. In addition there are dyspnea, especially on exertion, inertia and a tendency to chilliness. If the child passes the period of infancy, its growth is usually retarded.

On examination, the precordium is usually seen to be rather prominent and the cardiac impulse heaving and diffuse in character. In those cases in which the cyanosis is marked, the retinal vessels may be tortuous and their lumen irregular, being in some places very wide and in other places very narrow. A systolic thrill may be felt over the base of the heart to the left of the sternum. Percussion shows the transverse area of cardiac dulness to be increased, especially to the right of the sternum. On auscultation a loud, harsh systolic murmur may be perceptible over the whole heart but with its maximum intensity at the pulmonary area. The murmur may be localized or heard only at the pulmonary area. The second pulmonic sound is feeble or absent.

The differentiation of this murmur from other systolic murmurs heard at the pulmonary area has been considered under acquired pulmonary stenosis.

Patent Foramen Ovale.—During fetal life there is normally an opening in the interauricular septum, known as the foramen ovale. Shortly after birth this opening closes. In some instances, however, the opening persists and allows of the passage of blood from one auricle to the other (see Fig. 409); not infrequently the opening persists but does not allow of leakage (see Fig. 410). In the great majority of cases, in which after death a small probe can be passed through the foramen, the condition is not to be looked upon as abnormal.

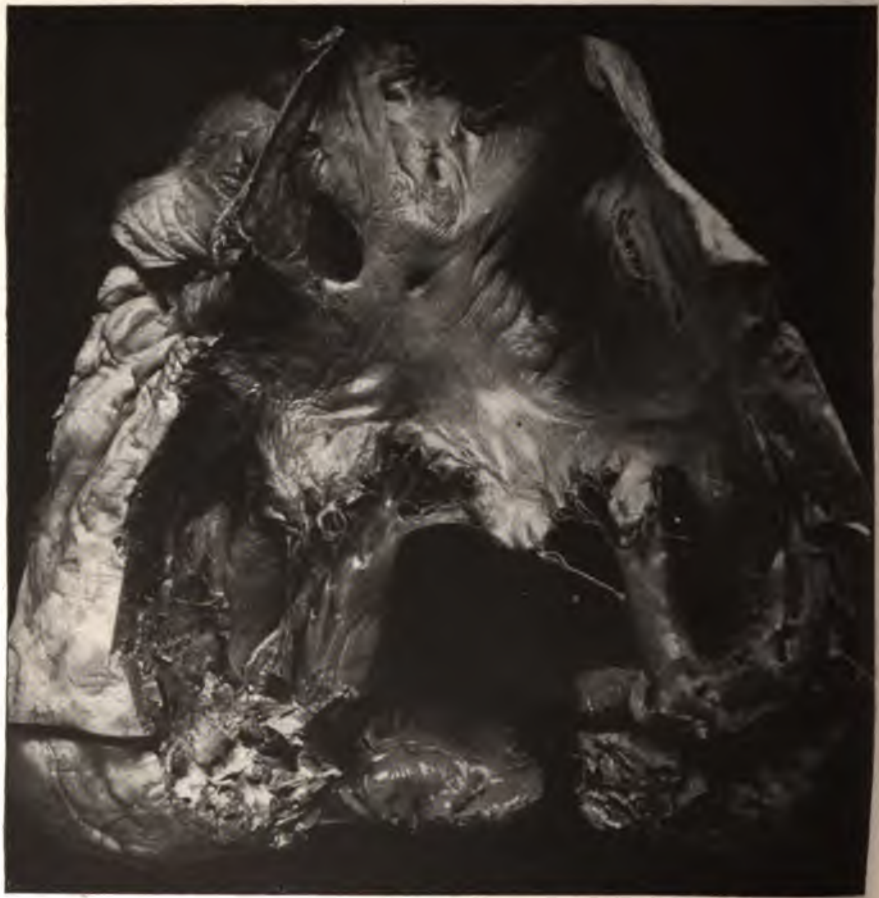
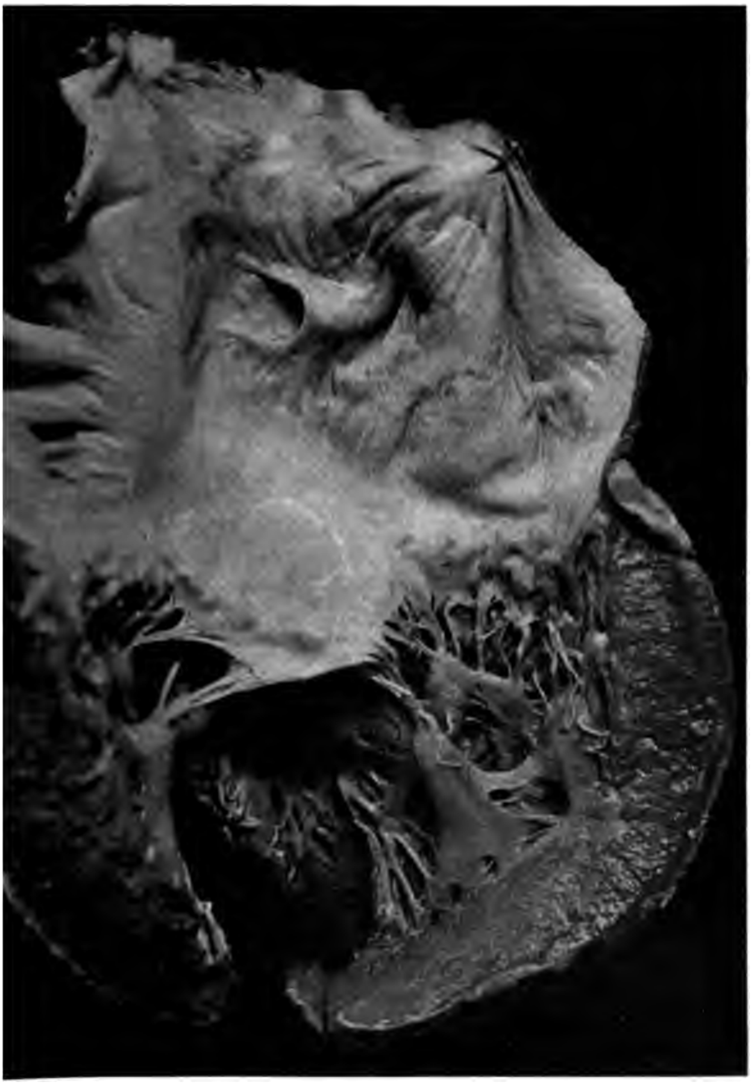


FIG. 409.—Patulous but incompetent foramen ovale. *(Pennsylvania Hospital.)*

If, however, the opening is widely dilated it may be considered as being anomalous and in such cases serious disturbances in the circulation are apt to occur. The fact that the channel runs obliquely through the auricular septum, and that, therefore, the openings on the two sides are not directly opposite to each other, favors competency. A demonstrable opening was recorded in only 86 cases among 8640 autopsies (0.9 per cent.) at the Philadelphia General Hospital (Norris).

Patency of the foramen ovale may occur alone but it is frequently associated with other defects and particularly with pulmonary stenosis.

ms and Physical Signs.—Small openings are not apt to give er signs or symptoms and even a wide opening (see Fig. 416) without manifesting itself during life. In a well-marked case



—Patulous foramen ovale. Heart of a woman aged seventy years, who died pneumonia, having advanced general arteriosclerosis. The illustration shows ore or less diffuse, mitral thickening, with contraction of the chordeæ tendineæ, hypertrophy of the left ventricle. The foramen ovale is quite large and patuing to its oblique course it was probably functionally competent.

toms may be those of congenital disease, namely, cyanosis, often paroxysmal in character, cold extremities, inertia, etc. urmur may be systolic, diastolic or presystolic in time and is

or undefended space, so-called because normally the septum here consists of two layers of endocardium. In the majority of instances this defect occurs in association with other anomalies. In 78 per cent. of Abbott's cases it appeared in combination with other defects; most commonly with pulmonary stenosis. In addition to causing serious alterations in the circulation, these perforations often become the site of an acute endocarditis (see Fig. 411). The effect of an imperforate septum upon the circulation and upon the heart depends on whether it exists alone or in association with other defects. When it occurs alone, it produces hypertrophy of both ventricles.

Symptoms and Physical Signs.—The symptoms will depend on whether the condition occurs as an isolated lesion or whether it is associated with other defects. In the latter case the predominant symptoms may be due to the associated lesion of which pulmonary stenosis is the commonest. When the lesion exists alone the symptoms are rarely marked and may consist of dyspnea and in some cases transient attacks of cyanosis on exertion.

This is one of the congenital defects in which the character of the murmur may enable one to arrive at a correct diagnosis. The murmur was first described by Roger in 1879 and is often referred to as the *brûit de Roger.* It is usually loud and harsh and best heard in the third interspace to the left of the sternum or to the left of the xiphoid cartilage. It is fixed and is not transmitted as is the case with organic valvular murmurs. The characteristic feature of the murmur is its length. It commences early in systole and is prolonged into diastole so that it obscures the second sound entirely. Roger in his original description states that "the murmur corresponds with a very extensive thrill which exactly coincides with it." At the base of the heart the second pulmonic sound is accentuated. The diagnosis rests on the hearing of a loud, continuous murmur with no interval between the systolic and diastolic portions to the left of the sternum.

Patent Ductus Arteriosus (Botalli).—The ductus arteriosus is an essential component of the fetal circulatory apparatus. It is a short thick trunk connecting the left branch of the pulmonary artery with the aorta and serves to carry the unaërated blood from the head and upper extremities to the descending aorta, and thence to the placenta. At birth it quickly ceases to be used and becomes impermeable about the third week. It is finally transformed into the ligamentum arteriosum. Just as in the case of the foramen ovale the duct may remain patent and as a result may or may not lead to circulatory disturbances. Patency of the duct may be the only anomaly present but more commonly it is associated with other defects, namely, pulmonary stenosis, transposition of the great vessels (see Fig. 412) or stenosis of the aorta (see Fig. 413). Among 106 instances of patency of the duct Abbott found but 19 in which the lesion occurred alone. Goodman[1] collected 71 cases, 34 of which were autopsied and the remainder were clinical observations. Recently Stoddard[2] has added 22 additional cases. A case of aneurismal dilatation of the ductus Botalli has been reported by Dry.[3]

Hypertrophy with or without dilatation of the right ventricle is a constant finding in adult cases.

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[1] *Univ. Pa. Med. Bull.*, December, 1910.

[2] *Arch. Int. Med.*, July, 1915.

[3] *Proc. Path. Soc. Phila.*, xix, 1917.

Symptoms and Physical Signs.—In uncomplicated cases the symptoms are, as a rule, not striking, and may be wanting entirely. Cyanosis and clubbing of the fingers, so commonly seen in other congenital affections, are unusual. Dyspnea and palpitation on exertion may occur.

The diagnosis depends, almost entirely, on the physical signs, of which the murmur is the most important. The murmur is produced by the rush of blood through the duct from the aorta to the pulmonary artery. Its point of maximum intensity is in the second left interspace. The murmur is usually loud, harsh, and of a churning or rasping quality. As in the case of imperforate ventricular septum the murmur is very long beginning in systole and continuing into diastole. It may be loudest at its commencement and gradually diminish or it may gradually become intense at the middle period and then wane. In other instances the murmur may be double or while continuous may have a systolic and a diastolic accentuation. The second pulmonic sound is usually much accen-

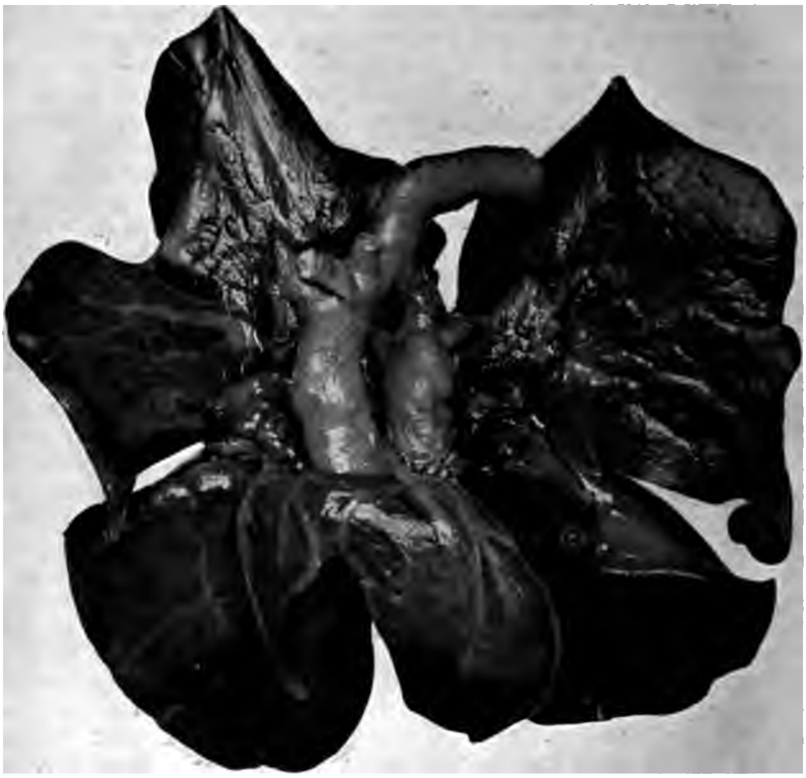

Fig. 412.—Patent ductus arteriosus. Transposition of the aorta and the pulmonary artery; patent ductus arteriosus; hypertrophy of the right ventricle. Patent foramen ovale; artial atelectasis of the right lung; general visceral congestion. (*A. G. Ellis.*)

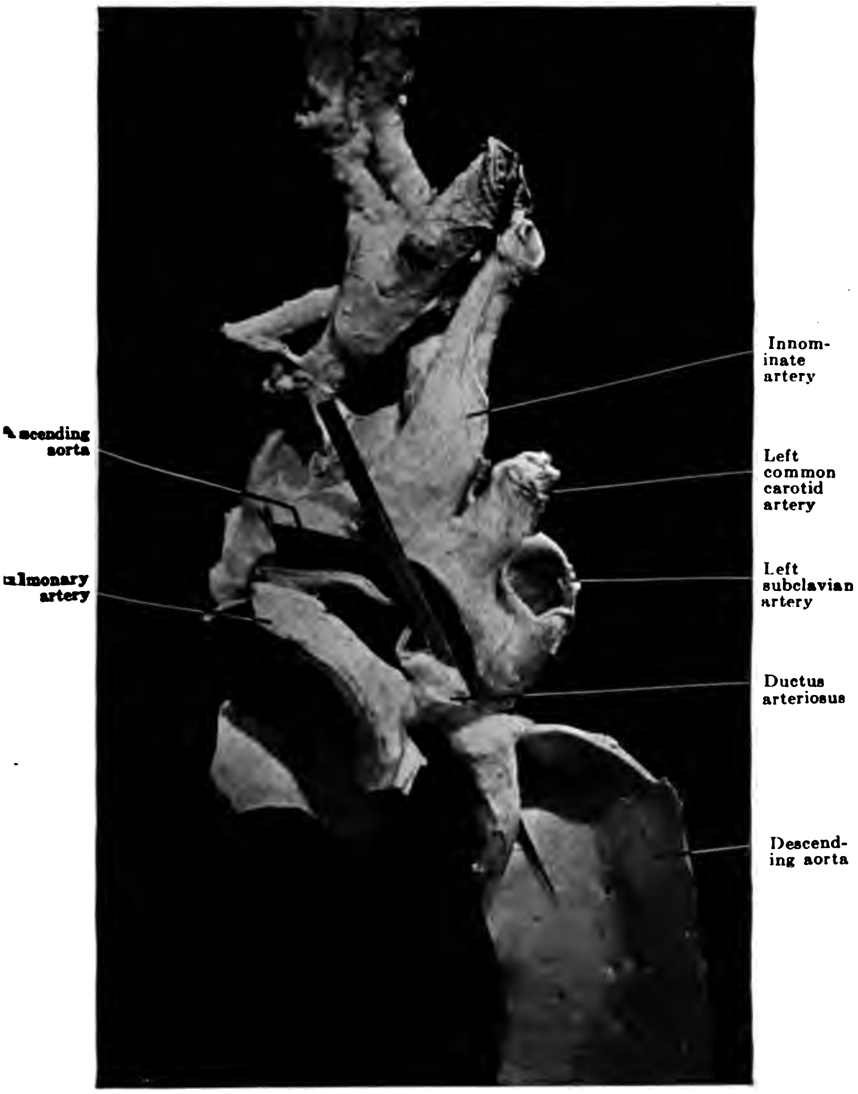

Fig. 413.—Congenital aortic stenosis with patency of the ductus arteriosus. Patho-
gic Notes: The aorta is very small, measuring at the arch only 4.5 cm. in diameter,
hile the pulmonary artery is twice this size. The innominate, left vertebral, and sub-
avian arteries are normal in position and size. Immediately at the junction of the lower
ge of the left subclavin artery and the aorta, the latter vessel shows a sudden and marked
nstriction, the external diameter of which measures 12 mm., while 3 cm. above and below
e constriction the aorta measures 2 and 2.5 cm. in diameter. On opening the aorta and
oking down toward the constriction the vessel appears to end in a smooth, rounded, blind
uch, but on closer inspection a pinhead-sized opening can be seen, through which a small
robe can be passed. (*Pennsylvania Hospital.*)

tuated, a point which serves to distinguish the murmur from that caused by pulmonary stenosis. The murmur is transmitted upward along the left margin of the sternum into the left carotid. It is also well heard posteriorly in the left interscapular region, being loudest during expiration.

A systolic thrill is often felt at the base of the heart to the left; the thrill may be felt throughout the cardiac cycle.

Gerhardt, in 1867, called attention to the presence of dilatation of the pulmonary artery in these cases. This is recognized by the presence of systolic pulsation in the second left interspace, due to forcible closure of the pulmonary valves, and an extension of dulness in the first second interspaces to the left of the sternum. Dilatation of the pulmonary artery may be determined also by the use of the fluoroscope or X-ray plate. In a case now under my observation the occurrence of a loud rasping murmur, best heard in the second left interspace, and the presence of a shadow in the X-ray plate indicating dilatation of the pulmonary artery, point toward a patent ductus arteriosus. The value of the X-rays in these cases, however, is not yet established. Stoddard[1] states that only two cases showing a dilated pulmonary artery with roentgenoscopy have been confirmed by autopsy.

Dextrocardia.—This term is applied to the condition in which the heart is on the right side instead of the left. Two varieties of dextrocardia are to be recognized: (1) *True dextrocardia* which is a congenital anomaly and is the result of transposition of the viscera. Complete transposition of the viscera, a condition in which the heart, stomach and spleen are on the right side and the liver on the left, is not excessively rare. Complete transposition of the viscera interferes in no way with the normal functions and is usually discovered accidentally during a routine examination. It has no clinical signifiance. Transposition of the heart alone may occur but is very rare (2 out of 412 congenital affections collected by Abbott). In such cases there may be a disarrangement of the great vessels with symptoms of congenital disease.

2. *Pseudo-dextrocardia* may be used to designate those cases in which the heart is displaced to the right but not transposed. It occurs whenever there is a large left-sided pleural effusion which pushes the heart and mediastinal contents to the right. Displacement of the heart to right is often very marked when there is present extensive fibroid disease of the right lung. In such cases the retraction of the fibroid tissue draws the heart and mediastinal contents to the right. The retraction and diminution in size of the right chest which ensues tends to emphasize the displacement of the heart (see Fig. 28).

Fishberg[2] has called attention to the frequency of acquired or pseudo-dextrocardia in cases of *tuberculosis*, especially in the advanced stage. He finds that the heart is not only displaced but slightly rotated on its vertical axis. This tends to bring the base of the heart closer to the chest wall and push or pull the apex away from it.

It is to be borne in mind that displacement of the heart is entirely different from transposition. In the latter the heart occupies the same position in the right chest that it normally does in the left. In cases of displacement the heart maintains its normal relationship but is pushed or pulled to the right although in cases of effusion the apex beat may be displaced upward. To speak of the apex beat being seen or felt in one of the interspaces to the right of the sternum in cases of displacement, is an error. What is mistaken for the apex beat is the pulsation of the right ventricle which has been pushed over by an effusion or pulled over, and at the same time exposed, by the retraction of the fibroid lung.

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[1] *Loc. cit.*

[2] *Arch. Int. Med.*, April, 1914.

CHAPTER XXIX

ANGINA PECTORIS

There is considerable uncertainty as to the proper classification of the various painful sensations centering about the precordium. There are two types concerning which most observers are in agreement: (1) True angina pectoris which is usually associated with disease of the coronary arteries and which, in the great majority of cases, is fatal. (2) Precordial pain which is due to disturbed function; is lacking either the intensity or the distribution of true anginal pain; is not associated with disease of the coronary arteries or the heart; and is never fatal. In this group may be placed those instances of heart pain occurring in neurotic, emotional women and in those addicted to the excessive use of tea, coffee or tobacco. Pain due to the excessive use of tobacco is not common. It may be very severe. Attacks of pain due to this cause are prolonged, as compared to true angina. Painful sensations arising under these circumstances are often referred to as pseudo-angina or angina pectoris vasomotoria.

In addition to these two groups, which are, as a rule, readily differentiated, there is a third group which may be termed incipient angina pectoris major. In this form of the disease there may be nothing more than a sense of oppression about the heart or if pain does occur it is not severe nor does it radiate. Many deny to these lighter attacks the name angina pectoris. There can be no question, however, that they are often the forerunners of true angina and that as time goes on the attacks tend to become more and more severe until they finally manifest all the characteristics of the major form of the disease.

Symptoms of incipient angina pectoris are especially apt to occur in patients suffering from one of two conditions, namely, arterial hypertension or aortitis, especially the syphilitic form of the disease. The importance of this group cannot be overestimated because a correct interpretation of the pain enables us to institute treatment which will either cure the trouble or at least mitigate the symptoms and prolong the patient's life.

ANGINA PECTORIS MAJOR

Etiology.—In the great majority of cases true angina pectoris is one of the manifestations of a general arterio-sclerosis, an organic heart lesion, or both; the etiological factors are, therefore, essentially the same.

The disease is encountered most frequently between the ages of forty and seventy, the highest incidence being in the fifth and sixth decades. When true angina occurs in an individual under forty years of age, syphilis is the most probable cause.

Although degenerative changes in the arteries occur in all walks of life and are often encountered in their most severe form among the working classes, angina pectoris is rarely encountered among these people.

It would seem that in addition to the arterial changes other factors are necessary, namely, worry and the strain and stress of high-pressure living. Certain it is that the vast majority of cases of angina pectoris major are met with among those engaged in pursuits entailing much responsibility and in whom the mental strain is great. Furthermore, in not a few instances such individuals increase their liability to the disease by reason of overindulgence in eating, in drinking and in the excessive use of tobacco.

It has been frequently commented that members of the medical profession seem to be especially liable to this affection. In a series of 268 cases Osler states that no less than 33 were physicians.

While angina pectoris does occur in women, it is relatively infrequent.

Occasionally families are encountered in which there is apparently a predisposition to arterio-sclerosis which terminates in a fatal attack of angina pectoris. In a family under the care of one of us (Norris) no less than five brothers and sisters have died in this way. The disease has been noted as occurring in the members of three successive generations.

Morbid Anatomy.—Hoover classifies the anatomical lesions in true angina as follows:

1. Isolated disease of the coronary arteries unassociated with any affection of the aorta, aortic valves, or systemic arteries.
2. Disease of the coronary arteries associated with systemic arterial disease.
3. Partial or complete occlusion of the lumen of the coronary arteries at their origin on account of disease of the aortic valves or disease of the root of the aorta. (Allbutt believes all cases of angina to be associated with disease of the aorta.)
4. Absence of disease of the vessels and heart, the vascular crises being due to arterial spasm, the result of vasomotor disturbances.

Finally, it should be mentioned that in some cases one of the coronaries may be blocked by a thrombus or embolus.

Arterio-sclerosis of the coronary arteries is by common consent accepted as the pathologic basis of anginal seizures. The radiation of pain to the chest, neck and arms is apparently a segmental phenomenon due to radiation and transference of pain to the periphery from those segments of the cord which receive impulses from the heart (third cervical to third dorsal) (see "Zones of Cutaneous Hyperesthesia," Figs. 42 and 43).

In seeking for an explanation as to the mechanism of the phenomena which characterizes angina pectoris the *intermittent claudication hypothesis* is most in favor. We know, for instance, that a restricted blood supply is a recognized cause of cramp in the muscles and that if the muscles are not properly irrigated and waste products removed, exhaustion with or without pain is likely to ensue. The most familiar clinical example of this is the condition known as intermittent claudication, in which, as a result of narrowing of the arteries of the legs, pain develops if the patient overexerts himself either by walking too fast or too far. In old people whose arteries have undergone degenerative changes, severe cramps in the legs or thighs are sometimes induced by walking; in other instances, the condition manifests itself by extreme fatigue of the leg muscles. So long as the patient keeps relatively quiet, the diseased vessels are capable of furnishing an adequate supply of blood, but if extra demands are made on the muscles more blood is needed and this the damaged arteries cannot supply.

The result is a local anemia with impairment or loss severe pain.

The constricting effect of diseased vessels as a ca with or without pain, in muscles during action w Allan Burns in 1809. In support of this contention a ligature be applied to a limb with moderate tigh admitted to the muscles for the performance of quiet action is attempted the muscles quickly become fat is apt to occur.

It is thus apparent that the vascular crisis whic pectoris has much in common with that occurring dication. If the function of the coronary arteries either as the result of partial occlusion of their openi an endarteritis the blood supply to the heart is limit sufficient providing undue demands are not made up

Furthermore, it is known that angina attacks ar by walking too fast, by walking against the wind or overexertion. In addition sudden emotion, partic cause a seizure. Whether because of exertion or an taxed by the suddenly increased blood-pressure, the supply the extra amount of blood demanded and as or even sudden death are produced.

In those instances in which the coronary arteries the only plausible explanation is that the anginal s spasm of the arteries. This hypothesis is supported or disturbance of function are produced by arterial of the body. Intermittent claudication, for instan to an endarteritis or to spasm of a healthy artery. are to be placed transient attacks of aphasia and tra the latter instance it is possible to see the spasm of means of the ophthalmoscope.

There are few who support the hypothesis th angina pectoris may be caused by neuralgia either in the gray matter of the cord or cardiac ganglia or result of a neuritis of the cardiac nerves.

Symptoms.—While an attack of angina pectoris the patient's life, it is a mistake to assume that a fa rule after a single or even two or three seizures. It that the attacks manifest themselves in varying de that seizures of true angina pectoris may occur at ir a long period of years.

Although angina pectoris major sooner or later e majority of cases, complete recovery has been note of severe attacks.

In considering the morbid anatomy of angina it w the majority of cases the condition was associated (aortic, coronary or general) and some form of o usually chronic myocarditis. An individual su vascular disease may either be free from symptoms be so slight as to pass unnoticed, and yet he may be of angina which terminates in sudden death. Su due to thrombosis or an embolus of one of the coron

In another and larger group, the initial attack may occur also without nonitory symptoms but the patient survives only to succumb in a sequent seizure. Such individuals may suffer from a number of icks which occur at irregular intervals and may extend over a period ears. In rare instances the primary paroxysm is followed by rapidly irring attacks which soon prove fatal.

By far the largest number of victims of the disease manifest some lence of their cardio-vascular disease prior to the appearance of angina in addition they are apt to suffer from symptoms which, although not ical of true angina, are at least "anginoid" in character. In such s there may be persistent arrhythmia and an ever-increasing suscepti- y to attacks of dyspnea, especially after exertion. Still more sig- ant is a feeling of tightness across the upper part of the chest or a e of pressure beneath the sternum after exercise or a full meal. These er feelings of tightness and distress in the chest may become increas- y painful, finally developing into the pain characteristic of true or or angina.

In most instances the anginal attack can be traced to a definite ting cause. The most frequent source is muscular exertion such as king briskly or running upstairs. Sudden emotion, especially anger, iot infrequently the cause of a seizure. In not a few instances ention of the stomach following a full meal precipitates an attack accounts for some of the cases of sudden death ascribed to acute gestion. Ingals and Meeker[1] have emphasized the fact that the i of angina is often centered in the epigastrium, or even over the er part of the abdomen, and that this should always be borne in mind ire committing oneself to a gastric origin of the trouble.

As the result of exertion or intense mental emotion, there develops denly in the upper chest a feeling of intense pressure, as though it e held in a vise, or there is pain sometimes agonizing in character, which ters in the substernal region and radiates to the left shoulder and rn the left arm to the elbow or wrist. The pain may extend down h arms; or it may be reflected to the neck and jaw; or it may be abdomi- Rarely it is felt in one testicle. Coincidently with the pain there great mental apprehension and a fear of impending death. The ient, if he has been walking, stands still or sits down at once and iains breathless and immovable until the attack has passed off. a rule, the seizure lasts from a few seconds to a minute or two. ring the attack there is usually marked pallor or an ashy gray appear- e of the face and there may be also profuse sweating.

Dyspnea, while not an essential feature of angina pectoris, sometimes urs during the attack. It may resemble asthma, the breathing being i wheezy character; in other cases acute pulmonary edema and the ectoration of thin blood-tinged fluid may occur. When respiratory nomena do occur, they are usually coincidents.

The attack may terminate in several ways: (1) It may be cut short sudden death; (2) the patient may pass away in syncope; (3) he may it; (4) the attack passes off leaving the patient with a feeling of exhaustion ing for a day or so; and (5) recovery from the seizure may be complete in hour or less time. With the subsidence of the attack there may be ctations, or the voiding of a considerable quantity of clear urine.

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[1] *Jour. Amer. Med. Assoc.*, April 6, 1918.

Occasionally patients with cardio-vascular disease suffer from a vascular crisis in which there are pallor, great weakness, and a fear of impending death, but no pain. Such attacks are referred to as *angina sine dolore.*

Physical Signs.—There is nothing distinctive in the physical findings during the height of the seizure; furthermore, the attack is usually so ephemeral and the symptoms so urgent there is little time for a detailed examination. There is very frequently arrhythmia but it is noteworthy that in many instances the heart action is slow and regular. Not infrequently the radial pulse on one side is smaller than the other.

Examination of the patient during the intervals usually shows that there are evidences of arterio-sclerosis and organic disease of the heart either in the form of a chronic valvular lesion or a chronic myocarditis or both.

Janeway attached great importance to the presence of *arterial hypertension* in the diagnosis of true angina. He believed that in the absence of any demonstrable anatomical change in the heart the occurrence of a blood-pressure of over 180 mm. may be held accountable for the seizure. The negative evidence, however, is not so convincing. Especially is this true in very severe attacks when the pressure may be low. During the attack the blood-pressure may rise or it may fall. With intense pain regularly following slight exertion, and pallor of the face during the attack, a low blood-pressure seems to augur a bad outlook (Janeway).

Diagnosis.—The recognition of true angina pectoris is not difficult if due consideration is given to the symptoms and the associated cardio-vascular lesions. Additional aids are furnished by the age, the sex and the occupation of the individual.

INCIPIENT ANGINA PECTORIS

In this form of the disease a sensation of substernal soreness or oppression or slight pain may be the only manifestation. These minor seizures may occur for a time and then cease entirely; or they may become increasingly severe and finally have all the features of angina major.

Etiology.—The etiological factors of incipient angina are essentially the same as in the major type of the disease. There are two conditions, however, which deserve special mention in considering these anginoid or milder attacks of pain, namely, syphilitic aortitis and arterial hypertension. And while we have already referred to this relationship, the importance of this group of cases cannot be overemphasized.

When the aorta becomes the seat of syphilitic disease, it is the first part of the vessel just beyond the aortic ring that, as a rule, is affected. The aortic valves are also very frequently involved. This lesion is quite apt to produce a feeling of soreness or oppression beneath the sternum or there may be distinct pain. Sometimes these symptoms are the precursors of true angina or aneurism.

Another very important group is comprised of those in whom a feeling of substernal distress or tension are associated with slight muscular exertion, such as walking briskly, climbing a stair or the indulging in any form of exertion following a meal. On the other hand, the symptoms may be due to emotion or excitement. Hypertension should always be thought of under these circumstances as patients with an abnormally high blood-pressure (200 to 250 mm.) very commonly suffer from some substernal distress after slight exertion or as the result of excitement. The hypertension may occur without there being any demonstrable lesion of either the vessels or heart. It is of itself, however, sufficient evidence that organic disease will sooner or later manifest itself. Under these circumstances the importance of the anginoid symptoms cannot be overestimated.

Morbid Anatomy.—The pathology of incipient angina is identical with that of the major form.

Symptoms.—The lighter attacks of angina, in the beginning, consist of nothing more than a transient sense of oppression or distress beneath the sternum which is neither sharply localized nor does it radiate. In some cases there may be slight pallor and faintness, but there is lacking the shock and fear of impending death which characterizes the major seizures. These mild attacks may cease entirely or they may recur for years. In not a few instances, however, the feeling of tightness is replaced by pain which gradually becomes worse, and more and more assumes the character of the pain encountered in true angina.

Physical Signs.—Physical examination of the heart and peripheral arteries may be entirely negative. But in many cases a careful examination of the cardio-vascular system will detect some change which will suggest the cause of the heart pain. There may be slight cardiac hypertrophy; or the presence of an aortic murmur, either systolic or diastolic, less frequently a mitral murmur; or there may be some thickening of the peripheral arteries.

In every case presenting anginoid symptoms a Wassermann test and blood-pressure estimation should be made. In addition, the retinal vessels should be inspected as they not infrequently will show sclerotic changes prior to their detection in the peripheral arteries.

Diagnosis.—While it is probably true that anginoid symptoms in some instances may be functional in origin, it is equally true that they are often indicative of serious organic trouble. It is not easy always to differentiate between the two types. The only safe rule to follow is to exclude carefully every possible organic lesion. Only when the blood-pressure is normal, the Wassermann test negative, and there is no discoverable cardiac or vascular lesion, is one justified in ascribing to the symptoms a functional origin.

ANGINA PECTORIS VASOMOTORIA

(Pseudo-angina, Mock, Spurious or False Angina)

These terms are applied to those instances of precordial pain due to vasomotor disturbances. Aside from the occurrence of pain, which, as a rule, has but a superficial resemblance to true angina, attacks of this nature have nothing in common with true angina of either the major or incipient type.

Etiology.—Vasomotor angina may occur at any age although rare before the twentieth year. It is encountered more frequently in females than males and is of relatively frequent occurrence in women who are emotional or neurotic. In common with other manifestations of an unstable nervous system, false angina may show itself in several successive generations. During the menopause vasomotor disturbances are common and not a few women during this period suffer from false angina.

A very common complaint is a feeling of pain or oppression just beneath the heart, usually associated with dyspeptic symptoms, especially flatulency. In the majority of cases the pain is due to false and not true angina. But it is to be borne in mind that gastric distention following a meal sometimes provokes an attack of true angina. The location and character of the pain will serve to distinguish the two conditions in most cases.

Precordial pain is not uncommon in those addicted to the excessive use of tea, coffee or tobacco. There are some who believe that nicotine poisoning is of itself a cause of true angina. The evidence on this point, however, is not convincing. Many of those who are subject to attacks of true angina are at the same time heavy smokers. It seems more likely that the excessive use of tobacco merely provokes an attack in an individual with a latent organic lesion rather than being the cause of the lesion.

Attacks of precordial pain, functional in origin, may follow one of the acute infectious diseases, notably influenza.

Morbid Anatomy.—In the great majority of the cases of false angina there is no morbid anatomy. The pain may be ascribed to "a disordered innervation of the vessels, peripheral or visceral, resulting in their contraction, causing an increased pressure of blood in the cavities of the heart, and a consequent embarrassed action with pain and dyspnea" (Powell). Furthermore, the disturbance of the vasomotor mechanism may manifest itself with cold, numb and blue extremities, or attacks of headache, neuralgia, etc.

Symptoms.—Subjects of this form of angina may have pain which is indistinguishable from that occurring in the organic form of the disease although it is rarely as severe nor does it, as a rule, radiate to the left arm. As a rule the pain in false angina is diffuse but it may have its point of greatest intensity beneath the heart as in cases of indigestion. In other instances the pain is sharp and shooting in character. This type of pain is not infrequent in those addicted to the use of tea, coffee, or tobacco. Instead of pain there may be a sense of oppression about the heart. Palpitation of the heart is commonly associated with the pain and may be the most annoying feature of the attack.

One of the striking features of true angina is the immobility of the patient during a seizure, whereas in this form there may be great restlessness, the patient moving or tossing about during the attack. In addition to the pain and palpitation there may be more or less dyspnea and a sense of air hunger in mock angina, in marked contrast to true angina in which the breath is held.

Precordial pain, functional in type, is usually sudden in onset and lasts but a brief time; a sense of oppression or dull pain may persist, however, for a half hour or longer. In very severe cases the patient may faint.

Usually the attacks occur at irregular intervals and are apt to follow overexcitement, excessive smoking or an attack of indigestion; in hysterical individuals there may be a number in one day. The occurrence of precordial pain is very apt to make the patient apprehensive that serious cardiac disease is present and this of itself tends to induce an attack. This form of angina is never fatal.

In considering the etiology of the affection it was pointed out that emotional or neurotic women were most subject to false angina; and that other evidences of vasomotor disturbance were common. Among the latter may be mentioned coldness and numbness of the extremities, attacks of migraine, neuralgia and flushes of heat. The patient is usually of spare build although plethoric women at the menopause are very susceptible to attacks of precordial pain.

Physical Signs.—Examination of patients subject to angina vasomotoria may elicit nothing abnormal in the heart; neither is there disease of the arteries. On the other hand the heart action may be very rapid and irregular. During an attack the pulse is often small and irregular or intermittent.

Diagnosis.—The distinction between this type of precordial pain and rue angina rests largely on the presence or absence of organic disease of he heart and blood-vessels. Furthermore, false angina is more common n neurotic women; it is encountered at an earlier age period; and the attack lacks, as a rule, most of the characteristic features of true angina.

Allbutt[1] has given a very clear description of mock or spurious angina. t is notable, he states, how obviously the heart is upset—palpitation, arrhythmias, sudden stoppages and accelerations, which are not characers of true angina. The so-called "heart pain" present in many, if not all, of these cases, the sense of distention of the whole upper chest, often with submammary pain and local hyperesthesia, are neither in seat nor in kind ike the pain of angina. In addition the breathing, far from being held as in angina, is panting; the bodily state is not awe-stricken but restless and agitated.

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[1] "Diseases of the Arteries, Including Angina Pectoris," vol. ii, 1915.

CHAPTER XXX

DISEASES OF THE AORTA

ACUTE AORTITIS

Etiology.—Acute inflammatory changes involving a portion or all of the aorta are undoubtedly of more frequent occurrence than is usually believed to be the case. Very often the lesion first reveals itself at the autopsy but in not an inconsiderable number the condition, during life, is either entirely overlooked or its presence is masked by preëxisting symptoms and physical signs. The most complete description of acute aortitis is given by Allbutt.[1]

Acute aortitis may occur as a complication or sequel of one of the acute infectious diseases, such as scarlet fever, smallpox, erysipelas, septicemia, influenza or typhoid fever. The importance of typhoid fever as an exciting cause of acute endarteritis has been emphasized by French observers and in this country by Thayer. In 52 cases of typhoid fever the aorta showed evidences of a recent endarteritis in 21 instances (Thayer). Broadbent states that in young children who have died of pneumonia, septicemia or some acute bacterial infection, he has frequently seen at the autopsy small, yellow and elevated patches in the aorta which he regarded as instances of acute degeneration resulting from the action of microörganisms or their toxins. According to Allbutt acute aortitis is not of infrequent occurrence in children suffering from acute rheumatic fever.

When it develops during the course of one of the acute infections, acute aortitis seems to be less severe and less apt to cause serious symptoms than when superimposed on a chronic aortitis. Some observers believe, at least in so far as clinical evidence is concerned, that in the great majority of cases the acute lesion develops only when the vessel has become the site of a chronic lesion. This is particularly true in gouty individuals whose vessels have become atheromatous. It may occur also in cases of chronic lead poisoning.

Since the discovery of the spirocheta pallida and the Wassermann reaction it has become clear that many cases which would formerly have been considered as simple acute aortitis are in reality instances of syphilitic aortitis.

Morbid Anatomy.—Acute aortitis is apt to arise during the course of any of the acute infections and pass away unnoticed. With the exception of syphilis the acutely inflamed aorta may show no changes to the naked eye or the changes may be so slight as to pass unnoticed unless carefully looked for. Appropriate staining methods will usually bring out very clearly the acute degenerative areas (Fig. 414). Occasionally the intima may be reddened or it may have lost its glistening, polished appearance as the result of a thin fibrinous exudate over the surface. The

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[1] "Diseases of the Arteries, Including Angina Pectoris," vol. ii, 1915.

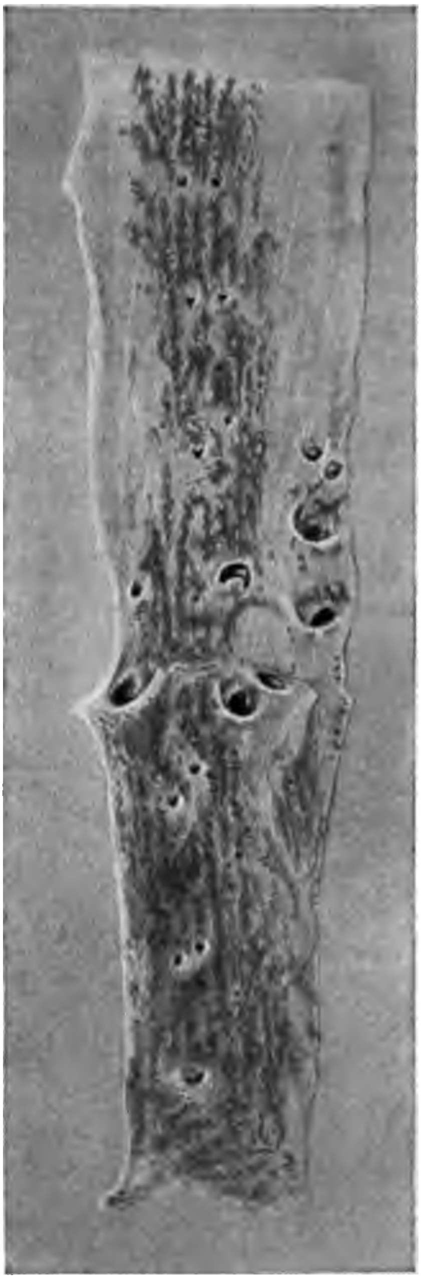

FIG. 414.—Acute aortitis. From a woman aged twenty-eight. Septicemia following abortion. Specimen stained concurrently with Sudan III and Scharlach R. Acute fatty degenerative areas deeply stained. Normal aorta should exhibit no more stain than shown in palest areas. (Jefferson Medical College Museum.)

first part of the aorta may be dilated and in most instances a recession to the normal usually takes place.

As usually seen, the degenerative changes are characterized by the presence of plaques ranging in size from that of a pinhead to a circle an inch in diameter. Not infrequently the plaques have coalesced. Their appearance varies with the stage of the process. At first they are slightly elevated, soft, pinkish-white or gray in color and opalescent; later they become firmer and yellowish in color. Microscopically the patches are seen to consist of an infiltration of the subendothelial tissue with round cells, spindle cells and stellate cells arranged in layers.

As the first part of the aorta bears the brunt of the infection, the orifices of the coronary arteries are sometimes partially, or entirely, occluded as the result of swelling of the adjacent tissues. It is this closure of the coronary vessels that gives rise to the anginoid pain.

Occasionally ulcerative endocarditis may extend and involve the root of the aorta. Rarely in cases of septicemia, abscesses may form between the external and middle coats of the artery and rupture inward.

Inasmuch as the vessel is often chronically diseased there may be noted also all grades of atheromatous degeneration.

Symptoms.—An individual suffering with an acute inflammation of the root of the aorta may be subject to attacks of difficulty in breathing and a sense of oppression in the chest. Pain is an important diagnostic symptom. Allbutt[1] describes the pain as having its "origin under the sternum, often at the junction of the first and second thirds of the bone, and a sense of constriction or compression, as if the chest were in a vise or the breast bone crushed inward by an iron bar. And this pain may come in any degree, from a transient sense of tightness or oppression about the upper sternum to utter torture." Sansom describes it as being "of a burning or tearing character, with a sensation of constriction or tension referred to the sternum." In most cases there is a tendency for the pain to radiate from the midsternal region into the left arm; occasionally into both arms, but rarely into the right alone. Among the symptoms of lesser importance are: fatigue, insomnia, vertigo, flatulence, nausea and sometimes vomiting. Fever is often present. The paroxysmal dyspnea and the substernal pain are the important features of acute aortitis.

Physical Signs.—Allbutt lays great stress on the physical signs, especially the percussion findings and the character of the aortic second sound.

Inspection.—There may be undue throbbing of the carotid arteries and in some cases *Potain's sign* may be noted. This sign consists of an abnormal upward displacement of the subclavian arteries so that they are seen or felt to beat in the hollow above the clavicles. This is due to dilatation of the aorta, causing its branches to be lifted to a higher plane. In the normal state the subclavian artery does not rise above the clavicle. McCrae[2] has emphasized the importance of inspection in these cases. Pulsation in the episternal notch is very common and if the aorta can be seen above the top of the sternum the diagnosis is almost definite. Pulsation should also be looked for in the first and second right and left interspaces, especially in the second right interspace.

[1] "Diseases of the Arteries, Including Angina Pectoris," vol. ii, 1915.

[2] *Medical Clinics of North America*, Sept., 1917.

Percussion.—The area of dulness will be found to occupy the manubrium, or at first the middle third of it, with an adjacent area of the second rib and third interspace one or two finger's-breadth to the right. The dulness about mid-manubrium is generally quite decisive. It may cross over a little to the left but is never so marked as on the right. Dulness may be elicited also posteriorly over the third and fourth dorsal vertebræ.

Auscultation.—Allbutt places great reliance on the quality of the aortic second sound but finds it difficult to describe. The term "clanging" he objects to. The quality of the sound is more tympanitic. Potain has termed it the *bruit de tabourka* from its resemblance to the sound produced by an Algerian drum, which is made of an earthen pot with a skin stretched over it. McCrae describes it as a musical bell-like sound which is distinctive and not heard in any other condition. In some cases there may be heard at the base of the heart a pericardial friction rub.

The aortic ring may become dilated or the valves may become inflamed as the result of extension from the aorta. If this occurs, a systolic or a diastolic murmur or both may be heard.

Diagnosis.—Acute aortitis may be confused with any of the conditions in which precordial pain is the dominant symptom.

Angina pectoris is apt to cause the most difficulty. In true angina the patient is usually a male, in good circumstances and beyond the middle period of life. There is also an associated cardiac lesion and evidences of arterio-sclerosis. Acute aortitis, on the other hand, may occur at any age period; the sexes are equally liable; and it also may occur in those of any walk of life. The pain in acute aortitis is usually localized behind the upper part of the sternum.

Syphilitic aortitis is characterized by symptoms similar to the acute inflammatory form of aortitis. The presence or absence of the Wassermann reaction would be the deciding factor.

SYPHILITIC AORTITIS

Etiology.—The discovery of the spirocheta pallida and the Wassermann reaction have made it clear that mesaortitis is often of syphilitic origin. Not only do a very high percentage of cases having lesions involving the aortic valves and root of the aorta show a positive Wassermann reaction, but in addition the spirochetæ can be demonstrated in the wall of the aorta. The susceptibility of the first part of the aorta to syphilitic infection has long been known although the definite proof is of comparatively recent origin. Larkin and Levy[1] found histological evidence of syphilitic aortitis in 17 of 19 cases giving a positive Wassermann reaction. In a series of 47 cases of aortic insufficiency Longcope[2] obtained a positive Wassermann reaction in 35, or 74.4 per cent. These results were further confirmed by finding in 7 cases at autopsy lesions typical of syphilitic mesaortitis and by the discovery in 3 cases of spirochetæ in the wall of the aorta. In a more recent article Longcope[3] expresses the belief that not less than three-fourths of all cases of aortic insufficiency are syphilitic in origin. Syphilitic aortitis also furnishes the explanation of the frequency of aneurisms of the arch of the aorta.

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[1] *New York State Jour. Med.*, December, 1915.

[2] Report of Ayer Clinical Laboratory, Pennsylvania Hospital, 1910, No. 6.

[3] *Arch. Int. Med.*, January, 1913.

The result of these comparatively recent additions to our knowledge of disease of the aortic valves and root of the aorta has been to establish a distinct group in which the etiology and pathology are essentially different from other lesions which present much the same symptoms and physical signs.

Syphilitic aortitis is encountered most frequently among those of early middle life. As in the case of other syphilitic manifestations, the condition occurs more often in males than in females. Among Negroes aortic insufficiency is very frequently syphilitic in origin. The condition may be encountered also in congenital syphilis. Rebaudi[1] in a study of the aortas of new-born children with spirochetæ in the principal organs found 13 of them abnormal. The lesions were similar to those of the aortitis of acquired syphilis in adults.

Morbid Anatomy.—The initial change consists of a small-celled infiltration of the media accompanied by an atrophy of both the muscular and elastic layers. The intima is not primarily involved but later undergoes proliferative thickening (Adami and Nichols). Eventually the fibrosis gives place to atheromatous changes.

Syphilitic aortitis is always confined to and often sharply localized in the arch of the aorta. In some instances it involves only the first part of the root of the aorta. The gross appearance is described by Longcope as follows: The margin of the sclerotic area is very abrupt, while the aorta beyond the lesion is quite smooth. In the most extensive areas are patches of thickening from 2 to 3 cm. in diameter. The central portion of these patches is elevated, gray and somewhat succulent in appearance, while the margins are yellowish and crinkled. The sclerosis, when marked, is characterized by an irregular, corrugated or crinkled thickening of the aortic wall, showing small pits and sometimes minute aneurismal dilatations. Often the bases of these small aneurisms are so thin that they transmit light. While calcification does occur the usual appearance is rather a rubbery, pliable thickening (Fig. 415). The aortic valves also show the same rubbery thickening when extensively involved and occasionally there are crescentic lines of whitish-yellow thickening.

The foregoing picture is essentially different from that encountered in rheumatic endocarditis in which the aorta is smooth and delicate in appearance. It also differs from the lesions encountered in general arterio-sclerosis. In the latter condition the atheromatous changes are not confined to the root of the aorta but involve the entire vessel and are often more marked in the thoracic and especially the abdominal aorta.

Symptoms.—In the majority of cases of syphilitic aortitis both the symptoms and physical signs are those of aortitic insufficiency. The importance of this fact cannot be overestimated. If the lesion is syphilitic in origin specific treatment will arrest its progress and if, as not infrequently happens, the disease is confined to the supra-aortic area the valves may be saved from damage.

"There will be fewer oversights in particular cases during life if the suspicion that an aortic lesion may be syphilitic is always with us. Even if we can elicit no evidence of an infection, the references from the story of the case, or from associated changes and relics elsewhere on the body, will generally bring us to a moral certainty. We know that a comparatively young man of otherwise healthy habits does not suffer from local proaching the subicteric. Psychic manifestations are also more frequent in the syphilitic type. In cases of this type a murmur is usually absent but a gradual weakening of the first sound, indicative of slight valvular involvement, may be noted. Grau[1] states that in the syphilitic cases the arterial phenomena are less marked than in the rheumatic cases. This is due to the fact that the entire aorta is often dilated and has lost its elasticity—hence the violent recoil of the blood current is modified.

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[1] *Monatsch. f. Geburtsh. u. Gyn.*, June, 1912.

In 23 cases studied by Grau the interval between the infection and the manifestation or aortitis was from six to forty-one years. Blumer believes that the period which elapses between the primary infection and the appearance of symptoms of aortitis averages eighteen years.

Whether the aortic insufficiency or the acute type of aortitis is syphilitic in origin will rest on the presence of a positive Wassermann reaction. In cases in which the aortic lesion occurs in combination with damage to the mitral and tricuspid valves, a rheumatic origin of the trouble is more than probable, but in isolated aortic lesions it is becoming more and more apparent that syphilis is the chief offender.

Physical Signs.—The physical findings are, in most cases, those encountered in aortic insufficiency. When the disease is confined to the supra-aortic area the physical signs are those of acute or chronic aortitis.

Diagnosis.—The early symptoms and signs of syphilitic aortitis are as follows: Dyspnea, which is often paroxysmal in character; pain, or a feeling of oppression in the precordium, in some instances the pain is felt under the short ribs, in others the pain is due to angina pectoris. More or less cardiac hypertrophy is present and the vessels of the neck show increased pulsation. Of the greatest importance is the presence of a positive Wassermann reaction and the signs of or the demonstration by the X-rays of dilatation of the aorta.

CHRONIC AORTITIS

(Arterio-sclerosis or Atheroma of the Aorta)

Etiology.—Arterio-sclerosis may be defined as a local or general thickening of the walls of the arterial tree together with a loss of elasticity due to an overgrowth of fibrous tissue and ultimately to the development of areas of degeneration (atheroma).

I. A general thickening and hardening of the arteries is an almost constant finding in people of advanced years. In the absence of any predisposing cause, the change begins to show itself, as a rule, at about the middle period of life and as time passes the sclerotic process tends to increase. While the entire arterial tree shows some evidence of fibrosis the change is not usually uniform. In one individual it is the arch of the aorta that shows the most advanced lesions; in another the abdominal aorta is most diseased; while in still another it is the cerebral arteries that have suffered most.

The frequency with which different parts of the arterial tree are most affected are: ascending aorta, the arch, descending aorta, abdominal aorta, iliacs, crurals, coronary arteries and cerebral arteries (Rokitansky).

II. In some individuals there is apparently an inherited predisposition to early arterial degeneration. The process occurs relatively early in life and frequently brings about a fatal termination at an age when the process should be in its incipiency. I recall one family in which both parents died as the result of arterio-sclerosis and three of the children succumbed to the same disease before attaining their fiftieth year.

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[1] *Zeit. f. Klin. Med.*, 1911, lxxii.

III. The male sex shows a higher percentage of cases than the female in the earlier years but subsequent to the menopause females are equally liable.

IV. The habits of the individual exercise a very potent influence on aggravating the condition. Among those in comfortable circumstances serious arterial changes are frequently encountered, especially if they have "lived high." The combination of overindulgence at the table, the immoderate use of alcohol and lack of healthful exercise are frequent causes of hastening the process. There are many cases in which overeating is the only factor (Osler).

V. Occupation plays a very important rôle in bringing about arterio-sclerosis. In one class of patients the work is entirely mental and attended with a constant strain on the nervous system. Thus the banker or business man in the fifth decade who is constantly engaged in ventures entailing great responsibility and long hours of work is suddenly stricken in his prime with a fatal apoplexy or his health is permanently impaired as the result of a degenerated heart muscle. An even more frequent result is a gradual failure of health as the result of associated changes in the kidney. The occurrence of advanced arterial changes in men of this class must be ascribed to their high-pressure methods of living as they are frequently abstemious in their habits and have suffered from neither syphilis nor gout.

The other extreme is the common laboring man who for years has followed a laborious occupation; has lived in insanitary places; has had a poor dietary; and has overindulged in alcohol. The most extreme cases of arterio-sclerosis I have encountered have been in individuals of this class. Many of them after the age of 60, possess arteries which certainly deserve the name "pipe-stem."

VI. Sooner or later in every case of arterio-sclerosis a constant accompaniment is a diseased kidney. It is not at all clear which is the antecedent trouble. Some observers consider that the cardio-vascular changes result from defective elimination; others, that the kidney becomes affected as a result of the arterial changes. In some cases, evidences of nephritis are first to appear; in others the kidney changes manifest themselves secondarily.

VII. Among the toxic causes lead and alcohol are the most important. Although Cabot discredits the influence of alcohol as a predisposing cause of arterio-sclerosis the prevailing opinion is, that either alone or in association with other factors, it cannot be disregarded.

VIII. Among the diseases which seem especially to predispose to arterial changes may be mentioned syphilis, gout, rheumatism, chronic arthritis and certain of the acute infections, notably typhoid fever.

Of more importance, probably, than any of the single etiological factors mentioned is *syphilis*. In so far as the aorta is concerned it is the most important factor as syphilitic arterio-sclerosis frequently takes the form of a mesaortitis. As a race, the Negro is especially prone to develop marked degenerative changes in the arteries as the result of a luetic infection. Syphilitic arteritis has been found to be a very common finding among the West Indian Negroes employed in the construction of the Panama Canal, while in this country the occurrence of aortic aneurism and aortic insufficiency among Negroes is frequently noted.

Morbid Anatomy.—In considering the etiology of arterio-sclerosis it was shown that alterations in the arteries might be brought about by a number of factors, and that by far the most frequent cause was a physiological deterioration incident to old age.

One of the penalities of advancing years is a gradual loss of elasticity of the arteries. This occurs in all walks of life irrespective of whether the individual has or has not been subject to some known exciting cause. In some, the change occurs earlier and becomes more marked than in others, possibly as the result of a hereditary predisposition; in others, the arterial changes are hastened as the result of disease, such as syphilis, gout, or typhoid fever; in still others, the degenerative change is delayed and never assumes serious proportions. In a histological study of the aorta Klotz has shown that beginning with about the thirty-fifth year, there are evidences of early changes in the vessel. The earliest manifestations consist of degenerative changes in the media; with advancing years these changes become more and more marked and as a result the vessel gradually loses its elasticity. That such a result should occur is not surprising when we recall the physiology of the circulation. With each systole of the left ventricle the aorta, already filled with blood, has forced into it a fresh supply. To accommodate this the vessel becomes slightly distended and then immediately contracts. In addition as the blood meets with resistance in the peripheral capillaries there is a recoil and as the blood is prevented from escaping backward by the closure of the semilunar valves, the aorta is again subjected to a strain. Still another factor to be considered is that the pressure is always increased by curvature or tortuosity of the blood-vessel. If then we consider that from 70 to 80 times each minute, every hour and every day the aorta is subjected to repeated dilatations and contractions it becomes apparent that there will come a time when the elasticity of the vessel loses its tone.

The type of arterio-sclerosis occurring in relatively young men, and which is brought about by an abnormally high blood-pressure resulting from working and living at high pressure, is analogous to the senile form in the method of its causation. One is a natural result; the other is, so to speak, artificially produced.

The gross appearance of the aorta when it has undergone senile atheromatous change is as follows: In the earlier stage the ascending portion and the arch are studded with round or oval, slightly elevated patches of a yellowish or yellowish-white color. These patches vary in size and number and in the thoracic aorta are especially marked at the orifices of the branches (see Fig. 181).

In the advanced stage of the disease the formation intima may be almost entirely obliterated by the formation of rough, calcareous plaques (see Fig. 417); in extreme cases the aorta may be reduced to a rigid, brittle tube (see Fig. 416). Occasionally a chip is whipped off one of the rough edges of a calcareous plaque and a small branch of one of the distal arteries is occluded. Gangrene of the foot and leg sometimes is produced in

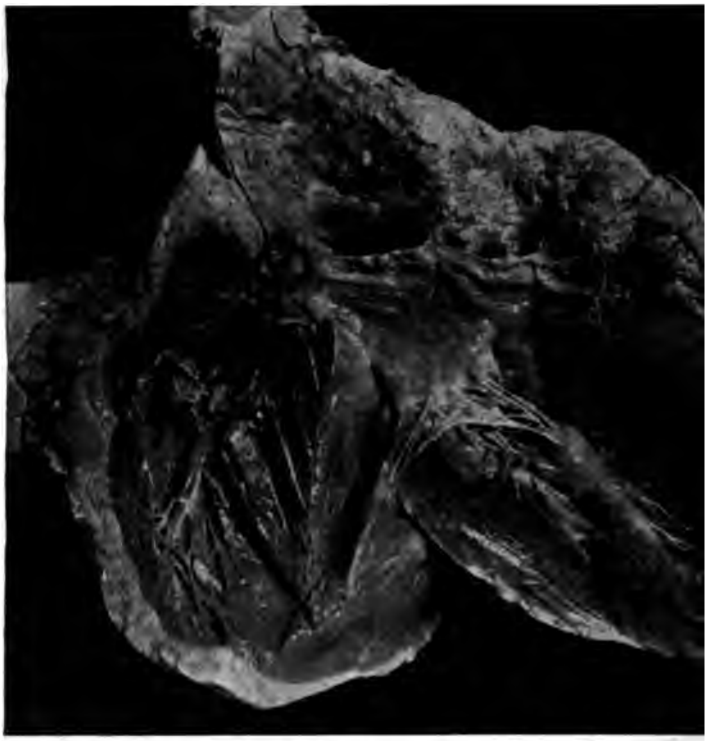

FIG. 416.—Calcification of the aorta. The entire aorta is one ha
thickened, calcareous mass, which fractures with an audible snap when t
The aortic valves share in this process of calcification. The wall of th
greatly thickened. The pericardium is thickened and the subpericardia
amount. *(Specimen from the Lankenau Hospital, Philadelphia.)*

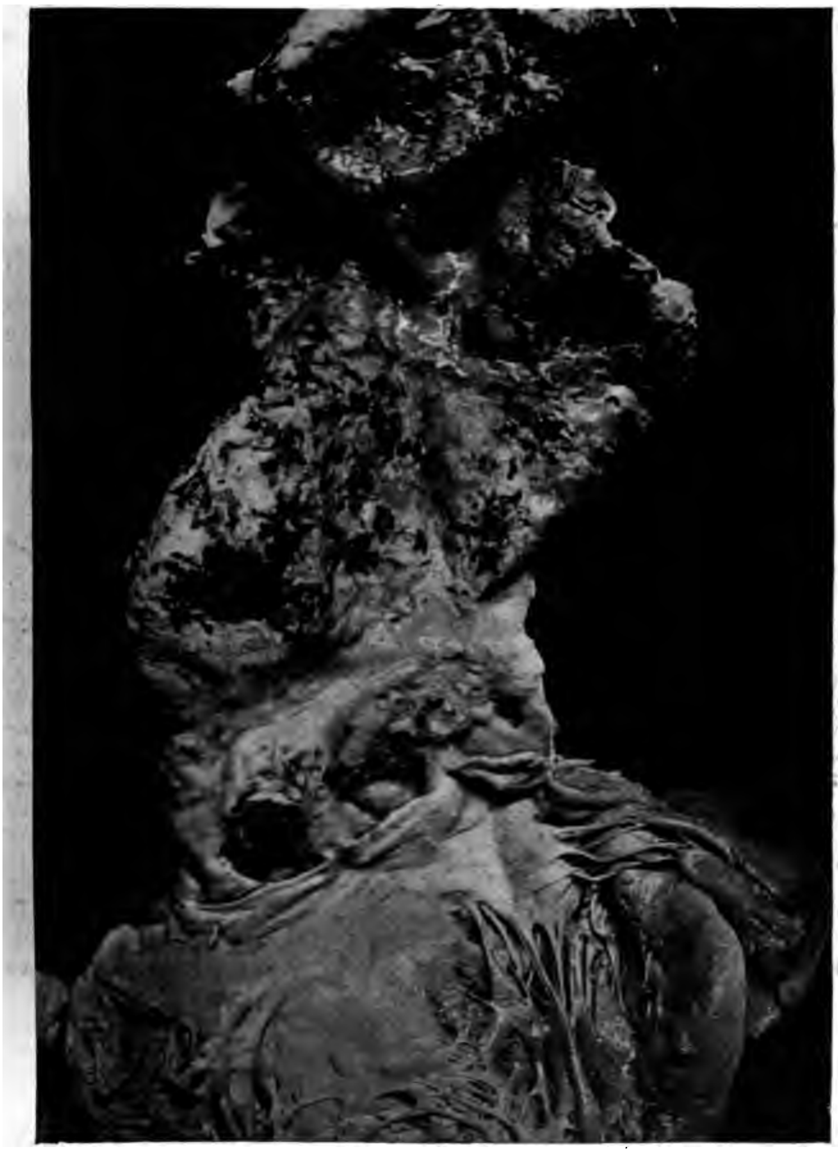

Fig. 417.—Calcification of the aorta. Specimen showing the left ventricular cavity. The aortic valves are thickened, slightly retracted, and contain calcareous deposits. The aorta is greatly thickened and its entire surface is covered with white and dark yellow, roughened and projecting calcareous plates. (Yellow being a relatively non-actinic color, these plates appear as black and brown in the reproduction.) The mitral valve is moderately thickened. (*Specimen from the Philadelphia Hospital.*)

namely, a primary change in the media followed sooner or later by degenerative changes in the intima.

The entire subject of aortic sclerosis is briefly described by Adami and Nicholls as follows: (1) In the vast majority of cases, if not in all, a weakness and giving away of the media is the primary anatomical lesion.

(2) There is a possibility that, as the result of a subacute proliferative intimitis, due to bacteria and their toxins, the thickening of the intima, by cutting off the nutrition of the inner layers of the media, may weaken that coat, and so cause a local dilatation of the aortic lumen, followed by a secondary and further thickening of the intima; but it is also possible that the infective endoaortitis which undoubtedly exists has no direct association with the general process here described, and that when, after typhoid and other infections, there develops a premature arterio-sclerosis, here, again, we deal with a primary sporadic degeneration of the media, set up by bacterial toxins.

3. The affection of the media may be either a primary degeneration without signs of preceding inflammation, or may be of inflammatory origin (as in syphilis).

4. The intimal change secondary to the medial degeneration has none of the features of an extension of the morbid process from the media, but is of a wholly different nature. It is primarily of hyperplastic type—a simple connective-tissue hyperplasia unaccompanied by the phenomena which we associate with inflammation.

Arterio-sclerotic changes in the aorta due to syphilis have been described under the heading, "Syphilitic Aortitis." In this type of the disease the puckered, rubbery appearance of the lesions is characteristic.

When the arterio-sclerosis is purely senile in origin the heart may show no hypertrophy or at best but slight enlargement. In the other forms of arterio-sclerosis extreme hypertrophy of the heart is a very common condition, especially in relatively young individuals with associated renal changes and an abnormally high blood-pressure.

Symptoms.—Atheroma of the thoracic aorta alone rarely manifests itself in the early stages either by symptoms or physical signs, unless the exciting cause is the spirocheta pallida. Occasionally, however, nonspecific sclerosis of the thoracic aorta, especially when the process is diffuse, is attended with a vascular crises. In cases of this type there may be substernal pain, which at times assumes the character of angina, palpitation of the heart and marked dyspnea. These symptoms are apt to be precipitated or increased by physical exertion. Attacks of this kind are analogous to those sometimes occurring in the abdomen as the result of diffuse atheroma of the abdominal aorta or its branches and in the arteries of the lower extremities, the latter attacks being known as intermittent claudication.

In the majority of cases of atheroma of the ascending portion and arch of the aorta the process is part of a general arterio-sclerosis and the symptoms are indicative of a general rather than a local process.

The early manifestations of arterio-sclerosis are as a rule indefinite and gradual in their development. Among the premonitory symptoms are attacks of indigestion, transient attacks of faintness, headaches, irritability of temper, defects of memory, exhaustion after mental or physical exertion and cramps in the muscles of leg. While in no way pathognomonic the occurrence of such symptoms in an individual approaching or just past early middle life, should lead to a thorough investigation of the cardio-vascular system.

The constitutional symptoms of arterio-sclerosis vary greatly with the general make-up of the individual. In some patients there is pallor, loss of weight and a loose flabby skin; in others the individual is stout and if of a gouty diathesis there may be a florid complexion.

The manifestations of arterio-sclerosis are not uniform. In some cases there is a gradual deterioration of health; in others the cardiac phenomena predominate; in another group the clinical picture is that of chronic nephritis; in still others the symptoms may be entirely cerebral in origin; finally, a chronic bronchitis and emphysema may stand out most prominently.

While cardiac hypertrophy is not always present, the increased work put upon the heart in overcoming the resistance in the stiffened arteries or abnormally high blood-pressure without marked arterial changes frequently lead to that condition. As in hypertrophy due to other causes dilatation of the heart may occur. If the patient is seen first at this stage the presence of dyspnea and cyanosis, together with the signs of broken compensation and a mitral murmur of relative insufficiency, will likely lead to a diagnosis of cardiac failure due to myocarditis or chronic valvular disease.

The association of sclerosis of the coronary arteries and angina pectoris is well known. Occasionally an obliterative endarteritis involving the coronary vessels, and without symptoms of angina, is the only lesion which will explain cases of sudden death.

When the kidneys are extensively involved, the predominant symptoms are those of a chronic contracted kidney. In such cases the daily output of urine is increased and nocturnal micturition is common. Pallor is marked and while there is puffiness about the eyes edema is not marked in other parts of the body. Examination of the urine will show varying traces of albumin and the presence of hyaline and finely granular casts. It is to be borne in mind that the albumin and casts may be intermittently present and that for varying periods the urine may show nothing more than an abnormally low specific gravity.

When the cerebral symptoms predominate, there may be attacks of vertigo or a transient hemiplegia or aphasia. The latter are usually of brief duration and while recovery is complete recurrences are apt to occur.

It is thus evident that the clinical picture of chronic aortitis varies according to whether the process is confined to the aorta alone or is associated with a lesion of the aortic valves, aneurism, nephritis or a general arterio-sclerosis. As an isolated lesion it often gives no evidence of its existence, attention being directed to the aorta through an attack of acute aortitis or what Dieulafoy terms borrowed symptoms, namely, those produced by hypertrophy of the heart, aortic insufficiency, aneurism or nephritis.

Physical Signs.—While it is the exceptional case of chronic aortitis that manifests itself by symptoms, a considerable proportion of cases can be recognized through the physical findings.

Inspection.—Although the sclerotic process may be confined to the thoracic aorta it is more commonly a part of a general process involving the entire arterial tree. In a well-marked case the temporal arteries are seen to stand out prominently and are often tortuous. The brachials are also prominent, often kinked and pulsate strongly. If the aorta is dilated, a valuable diagnostic sign is pulsation in the suprasternal notch. In addition the right subclavian artery may be seen to pulsate above the clavicle, being lifted up as a result of the dilatation of the aorta. Arcus senilis is often noted in those of advanced years and at one time was considered as being an indication of sclerosis. The condition is due to colloidal degeneration of the superficial layers of the cornea. It is not infrequently seen in those relatively young and is quite common in Negroes.

Inasmuch as hypertrophy of the heart is so frequently associated with arterio-sclerosis, the cardiac impulse may be forcible and heaving in character with the apex beat displaced downward and to the left. Hypertrophy of the heart furnishes valuable evidence as to which portion of the arterial system is diseased or at least most involved. Thus arterio-sclerosis leads to a hypertrophy of the left ventricle only when the splanchnic arteries or the aorta above the diaphragm are highly diseased. The hypertonicity or arterio-sclerosis of the remaining vascular districts does not appear to exert this influence (T. C. Janeway).

If the renal symptoms predominate, pallor is apt to be a marked feature and there may be evidences of edema. The latter is usually slight; it may manifest itself by puffiness beneath the eyes and by slight swelling of the feet and tissues over the front of the tibia (pretibial edema).

There are two invaluable aids in determining the existence and degree of severity of arterio-sclerosis, namely, direct inspection of the eye grounds with the *ophthalmoscope* and the use of the *sphygmomanometer*. Prior to the introduction and perfection of these methods of diagnosis the early stages of arterio-sclerosis usually passed unnoticed and the condition was not recognized as such until the symptoms were well marked and the arteries had become both visibly and palpably thickened. Once the disease has become well advanced, there is little that can be done; on the other hand, if it is detected in its incipiency, the process frequently can be arrested. It is for this reason that the slightest of untoward symptoms occurring in individuals approaching early middle life should excite suspicion. Some observers advise that periodic examinations of the urine and blood-pressure be made in individuals of or approaching middle life in order to detect the degenerative changes in their incipiency.

It is to be deplored that the ophthalmoscope is not employed more generally by the internist who, if he cannot expect to become an expert with the instrument, can at least acquire sufficient skill to distinguish an abnormal from a normal eye ground.

The relation of the *retinal vessels* to arterio-sclerosis and nephritis is so important that even a suspicion of either should lead to an ophthalmoscopic examination. Largely as a result of the studies of Marcus Gunn it has been shown that the retinal vessels present a characteristic appearance in arterio-sclerosis and that alterations in these vessels often can be detected prior to changes elsewhere in the arterial system.

Retinal arteries which have undergone sclerotic changes show undue tortuosity, alterations in their size and caliber and also present a beaded appearance. In addition whitish stripes are seen in the arterial wall and there is a loss of translucency so that it is impossible to see, as is possible in the normal state, through the artery an underlying vein at the point of crossing.

The retinal veins are also tortuous and show alternate contractions and dilatations; and of even more importance is the indentation of the vein and impeding of the venous circulation where a diseased artery crosses it.

The retina itself may be slightly edematous, grayish opaque or show the presence of linear extravasations of blood along the course of the vessels or scattered roundish infiltrations over the fundus.

De Schweinitz states that the above-described changes may be the forerunner of sclerosis of the cerebral arteries and the only indication that such a condition is present.

Prior to the introduction of the sphygmomanometer the question of whether arterial tension was increased or not was determined largely by the sense of touch. We have all learned to appreciate how fallacious such observations are and how frequently a supposedly high tension is in reality normal or *vice versa.*

The determination of what constitutes a normal tension standard for the different age periods is somewhat arbitrary. Woley[1] gives the following results:

Age	Systolic blood pressure		
	High	Low	Average
15–30 years	141	103	122
30–40 years	143	107	127
40–50 years	146	113	130
50–60 years	149	115	132
60–65 years	153	120	138

The diastolic pressure is from 20 to 40 mm. lower than the systolic. In women the systolic pressure is from 8 to 10 mm. lower than in men at corresponding age periods. It is to be borne in mind that the pressure may be raised abnormally as the result of physical exertion, mental activity and, especially, excitement.

The fact that the blood-pressure tends to rise with advancing years must not be lost sight of. A systolic pressure of 150 mm. may be entirely consistent with good health in an individual of sixty years of age; the same pressure at thirty would be of the most serious import.

A constant systolic pressure of, or above, 160 mm., or a diastolic pressure of or above 100 mm. Hg. is pathologic at any age. *The diastolic pressure is a more active criterion of the actual average pressure than is the systolic, since it is much less subject to functional or emotional lability.*

While thickening of the superficial arteries, high blood-pressure, enlargement of the left ventricle and a ringing second aortic sound form a frequent combination in arterio-sclerosis, it not infrequently happens that the pressure is not abnormally raised. This may be due to poor nutrition, cardiac weakness, or degenerative changes in the arterioles.

Whenever the systolic pressure is markedly high, 200 mm. or over, there is always a strong probability that the kidneys are diseased, al-

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[1] *Jour. Am. Med. Assoc.,* 1910, iv, 121.

though among the well-to-do exceedingly high pre
and may occur without evident arterial thickenin
this type are referred to as instances of essentia
discussed more in detail in another place. It is i
that the sphygmomanometer is most serviceable.

Percussion.—Hypertrophy of the heart is indi
of the cardiac dulness both to the right and left.
will be an increase in the area of dulness due to
At the level of the second costal cartilage the un
gives rise to a transverse area of dulness from 2 t
in width. If, therefore, the dulness extends beyon
to the right of the sternum, dilatation of the aort

Auscultation.—The most common auscultato
chronic aortitis are the presence of a systolic mur
cartilage and a sharply accentuated, high-pitche
A systolic murmur at this area is a very commo
quently attributed to stenosis of the aortic outlet.
more common mistake in the diagnosis of cardiac
fact that aortic stenosis is recognized as the ra
(see p. 743).

A *systolic murmur* independent of disease of
be caused by changes in the first part of the aor
dilated or its inner surface may be roughened by
artery may be converted into a narrow, rigid tub
dilated, the blood current may be thrown into sw
murmur; if, on the other hand, the inner surface o
or the artery is rigid the propulsion of the blood
ened surface or through the rigid tube may also pr
in time. The conditions just mentioned are ver
part of a general arterio-sclerosis, hence the fre
Of almost equal importance is the sharply accent
of the second aortic sound.

In a much smaller proportion of cases a diast
When this is the case the lesion is usually syphili
mur may be produced in one of two ways: (1) A
extend and involve the aortic valves, thus giv
valvular lesion; (2) the aorta may become dilate
ortitis and in this way the aortic ring may becom
being any actual disease of the leaflets themselve

ESSENTIAL HYPERTENSIO

Under this heading is included that group of c
tial feature and earliest manifestation is a high
recognition of this condition is important as it is
chronic interstitial nephritis or arteriosclerosis.
been used to designate it. By some it has been
albuminuric stage of Bright's disease; by other
arteriosclerosis; by still others as the presclerotic
By Theodore Janeway the condition was referre
tensive cardiovascular disease and of recent year
introduced by Sir Clifford Allbutt, has gained fav

The cause of essential hypertension is, as yet, unknown, and none of the explanations so far offered are satisfactory. Among these may be mentioned: changes in metabolism, absorption of toxic material from the intestines, and focal infections. A possible cause is some perversion of secretion of the endocrine glands.

Individuals subject to essential hypertension usually first show symptoms of the disorder in late middle life. The condition is, however, often discovered accidentally in the course of a routine examination. In such instances there may be no symptoms whatever, other than the high tension, of which the patient is unconscious. For the most part individuals subject to this disorder are stout, plethoric and of a ruddy color. Although they usually indulge freely in the pleasures of the table they are not often addicted to overindulgence with alcohol. Men are said to be affected more often than women but it is certainly true that many women, especially those at or about the climacteric, develop a high blood pressure. Although essential hypertension is first seen in the great majority of cases, between the ages of forty and sixty it may be encountered in children or in those of advanced years. In regard to the latter group, however, it may be said that the hypertension may have been present for years and gone unrecognized.

When patients subject to this disorder seek medical advice they usually complain of a more or less persistent headache, dyspnea on exertion, palpitation, precordial distress or anginoid pain, headache, transient attacks of dizziness, a feeling of malaise or neurasthenic symptoms. In not a few instances the first evidence of trouble is some disturbance of vision due to a retinal hemorrhage. I have such an example of the disease under observation at the present time. This patient is a stout, plethoric man of about thirty-eight years of age who, because of failing vision in one eye, consulted an ophthalmologist who found a retinal hemorrhage. A few months later he had another hemorrhage. Aside from a high blood pressure he has no discoverable trouble. In another case the discovery of a retinal hemorrhage seven years ago led to an extremely gloomy prognosis based on the belief that the man had a chronic kidney lesion. Aside from a blood pressure of about 220 his condition remains unchanged.

The examination usually reveals, in addition to the high arterial pressure, some hypertrophy of the heart and, if the case is of long standing, tortuosity and some thickening of the peripheral arteries. Examination of the eye-grounds is important. In high tension cases there will be an indentation of the veins at the arterial crossings; tortuosity of the arteries; irregularity in the lumen of the arteries; central light reflex (a bright glistening streak along the center of the arterial wall); and retinal hemorrhages. An examination of the eye-grounds often reveals the condition before it is otherwise suspected.

A clear understanding of the urinary findings is essential in order to avoid the error of mistaking the condition for chronic nephritis. In essential hypertension the specific gravity of the urine ranges from 1010 to 1030. There may be a trace of albumen but commonly there is no evidence of albuminuria whatever; a few hyaline casts may or may not be present. More important still is the fact that the functional tests used to determine the permeability of the kidney show no abnormality. The elimination of phenolsulphonephthalein is normal and the figures for blood urea nitrogen are within normal limits. In contrast with these findings are those found in cases of chronic nephritis. In the latter condition there is polyuria, a constantly low specific gravity, a relatively large amount of albumen, and numerous casts. In addition the functional tests will show a marked reduction of the kidney function. Meara[1] attributes the trace of albumen in cases of hypertension to arteriosclerosis in the kidney and the heavy trace of albumen to passive congestion in the kidney.

It is quite possible that in some cases of chronic nephritis the initial stage may have been an essential hypertension but in the great majority of cases the termination of the disease is either the result of cardiac failure or an arterial accident. As already stated the heart is usually hypertrophied. This is brought about by increased work due to the high arterial tension. Cardiac failure may be brought about finally by inability of the heart to work effectively against the high pressure or because sclerotic changes in the coronary arteries affect the heart muscle. When this occurs the signs and symptoms of failing compensation manifest themselves. Symptoms referable to the heart are usually the first to indicate that the cardiovascular system has reached its limit.

Symptoms referable to the arteries themselves are usually cerebral in origin. Then there may be a rupture or thrombosis of one of the middle cerebral arteries resulting in a hemiplegia or aphasia. In other instances disturbances in the cerebral circulation may be transient in character. The memory becomes treacherous and the patient momentarily forgets his name, or there may be transient aphasia or confusion in the choice of words. There is often an inability to concentrate the mind on any task and in older people there may be a frank mental deterioration.

Among other disturbances which may occur may be mentioned pulmonary edema, paroxysmal dyspnea or tachycardia and attacks of severe anginal pain.

Essential hypertension may exist for years without any apparent inconvenience to the patient, in spite of a tremendously high arterial pressure. In my experience this seems to be especially true of women. Hopkins who has studied the subject carefully believes that essential hypertension occurs almost exclusively in women and that what is termed essential hypertension in men is in reality part of the complex of a chronic nephritis.

Among 100 cases observed by Rapplege[2] there were thirteen deaths, ten of which were associated either with the cardiovascular system or an acute infection.

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[1] *Medical Clinics of North America*, July, 1918.
[2] *Boston Med. and Surg. Jour.*, Oct. 3, 1918.

CHAPTER XXXI

ANEURISM OF THE THORACIC AORTA

n aneurism may be defined as any circumscribed or localized expan- or dilatation of the lumen of an artery.

the wall of the distended portion is composed of all or any of the s of the artery, the aneurism is called "true"; if the sac in part or in ntirety is formed by surrounding tissues or a newly formed fibrous stment, the condition is spoken of as a "false" aneurism.

n aneurism is almost invariably secondary to some disease of the y, such as atheromatous degeneration or syphilis. Occasionally an rism may arise in a healthy artery as the result of a sudden straining e wall whereby the intima is ruptured; as the result of a wound; or as esult of the wall becoming eroded by the extension of an inflamma- process from some adjacent structure. Syphilis, however, is to be ed upon as the most important factor in the causation of aneurism e thoracic aorta.

. *Dilatation of the Aorta.*—A slight enlargement of the lumen of the a is a relatively common condition. It constitutes what may be ed an incipient form of the disease and is very commonly overlooked ng life.

ilatation of the aorta may range from a slight increase in the diame- f the lumen of the vessel up to an extreme degree of distention. It is with most frequently in the ascending portion of the arch. In this tion the vessel presents a fusiform or cylindrical dilatation, the diame- eing increased from the normal 5 to 5½ cm. to from 6 to 9 cm. or even e.

. *Saccular Aneurism.*—When one speaks of an aneurism of the arch e aorta it is the saccular variety that is meant in the great majority of s. It is this type that is defined as being a pulsating tumor contain- blood in direct communication with the interior of the artery. It is ed by the bulging or giving away of a portion of the arterial wall which been weakened by disease or, rarely, by injury. It differs from dilata- of the aorta in that it is due to a circumscribed bulging at one point not to a stretching or distention of the entire circumference of the ry.

. *Dissecting Aneurism.*—Instead of a localized bulging at the point re the intima is destroyed by disease, the blood may tear its way be- n the intima and media for some distance giving rise to what is known dissecting aneurism. At some point farther along the course of the ry it may again rupture through the media and communicate with the rior (see Fig. 172).

. *Arterio-venous Aneurism.*—This form is rare in the aorta. Occa- ally, however, the aneurismal sac may rupture into the superior vena or pulmonary artery, constituting what is known as an aneurismal x. Instead of rupturing directly into the vein there may be a rupture of the aortic aneurism with the formation of a hematoma or false aneurism; later rupture into the vein may occur. When a false aneurism lies between the opening in the artery and that in the vein, the condition is spoken of as a varicose aneurism.

5. *False Aneurism.*—If all three coats of the artery are torn or destroyed by disease, the blood escapes and forms a hematoma. This may be walled off by the surrounding structures or, if gradually formed, a fibrous envelope may form. A false aneurism may be caused by rupture of the wall of a diseased artery or the artery may be healthy, the opening being caused by a wound or by erosion as the result of disease in an adjacent structure.

Etiology.—The cause of an aneurism is always to be sought in a primary disease of the vessel wall, which weakens its resistance to the blood-pressure. With the possible exception of trauma, the same factors which cause arterio-sclerosis also favor the origin of aneurism.

While any of the acute infectious diseases may cause arterial degeneration, such an occurrence is not common except in *syphilis.* As a predisposing factor in the causation of aneurism syphilis easily holds first place. It has always been acknowledged that the rôle played by lues was an important one. Now that we have come to recognize the existence of a syphilitic aortitis the percentage of cases due to syphilis has materially increased and at the present time it is estimated that from 70 to 85 per cent. of all aortic aneurisms are to be attributed to this cause. When an aneurism occurs in an individual under forty it is almost certain that a luetic infection is the cause.

Occasionally, certain of the other infections such as pneumonia and erysipelas may produce a localized mesaortitis, thus leading to a weakening of the arterial wall. Or an endocarditis occurring in the course of acute rheumatic fever, pneumonia or septicemia may extend to the aorta and as the result of inflammatory changes in the intima weaken the wall of the vessel.

While a very marked degree of arterial degeneration may be caused by lead, alcohol, tobacco, gout and chronic nephritis, aneurism is not of very frequent occurrence as the result of these poisons.

Next to the presence of an aortitis the most important factor in the production of an aneurism is *internal strain.* If the vessel wall has become markedly diseased it may be unable to resist the strain of a normal blood-pressure. In most cases of aneurism, however, the artery has been subjected to a severe strain such as occurs with sudden or violent muscular effort. This is indicated by the fact that a very high percentage of the cases having an aneurism have followed an occupation which demanded great muscular exertion, either prolonged or sudden. Thus stevedores, iron workers, draymen, soldiers and sailors are especially liable to the disease. Furthermore, the aneurism is most apt to develop in the active periods of life when the muscular strength and activity of the individual are at their highest point. Any sudden exertion, such as running upstairs, straining at stool or lifting is apt to produce a sudden rise in the blood-pressure and if there is present a patch of mesaortitis this sudden strain may cause the intima over the diseased area to split.

Passive dilatation of the aorta may be produced by persistent arterial hypertension such as occurs in chronic Bright's disease. In this way the diameter of the ascending portion of the arch may be increased up to 8 to 9 cm.

Among the rarer causes of aneurism may be mentioned *infective emboli*. In the large arteries septic emboli may lodge in the vasa-vasorum and produce a point of suppuration; later the overlying intima ruptures and in this way an aneurism may be formed. Other explanations of these mycotic aneurisms are that there may be an extension of an inflammatory process from a neighboring infectious process; that there may be a metastatic deposition upon the intima from a valvular endocarditis; and that the surface of the intima may become infected from a distant focus.

A violent blow on the chest or the jar of a fall may fracture the intima and produce a dissecting aneurism. Finally, an aneurism may be caused by the wall becoming weakened as the result of some external injury such as a bullet or knife wound or by disease of an adjacent organ.

It is evident from the above facts that aneurism is practically always secondary to arterio-sclerosis of the aorta; that syphilis is the most common cause of the arterial degeneration; and that, in addition to disease of the vessel, arterial strain, usually suddenly produced, is an essential factor.

For the reasons just given aneurism is usually encountered between the ages of thirty and fifty when muscular activity is at its height. Aneurism may occur at any age but is unusual in the very young and the very old. Typical aneurisms have been noted, however, in children three or four years of age; commonly there is a history of, or the evidences of, congenital syphilis.

Men are more frequently affected than women—in the proportion of about 5 to 1. This is due to the fact that men are more often infected with syphilis and are in addition engaged in laborious occupations. The incidence of aneurism among Negroes is high. Thus the Charity Hospital, of New Orleans, for the year 1917 had admitted to its wards 25 cases of aortic aneurism of which 22 were colored and 3 white. In the vast majority of Negroes suffering from aneurism syphilis is the cause. Powell[1] states that among the natives of India, the occurrence of thoracic aneurism is very common, and is one of the most frequent causes of sudden death. In the post-mortem room aneurism of the thoracic aorta is encountered in less than 1 per cent. of cases. Lemann[2] found 1 in 300, Osler[3] 1 in 100 and Borowsky[4] 66 among 19,646 autopsies (0.34 per cent.).

Morbid Anatomy.—Except in case of trauma, which is rare, the first step toward the formation of an aneurism is the development of an endarteritis; and particularly that form which is caused by syphilis (see Fig. 415). Chronic endarteritis which terminates in widespread atheromatous changes of the aorta is of common occurrence without aneurism. On the other hand, circumscribed or focal lesions, such as occur in syphilis, are extremely liable to lead to dilatation or bulging of the affected part of the vessel wall.

As a rule the aneurism is a late manifestation of syphilis. Winternitz[5] m may attain. An aneurism differs from other tumors in that it is le and it is to this fact that its destructive ability is probably due. ly does the tumor gradually increase in size but in addition the with which it is brought into contact are subjected to an incessant

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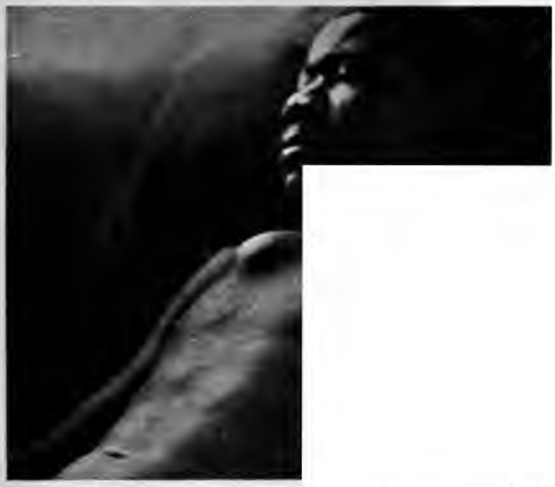
[1] *Proc. Royal Society of Medicine*, 1911, v, 186.

[2] *Am. Jour. Med. Sc.*, August, 1916.

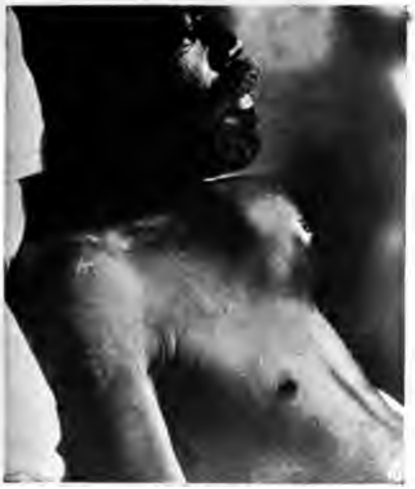
[3] Schorstein Lectures.

[4] Inaugural Dissertation, Breslau, 1910.

[5] *Bull. Johns Hopkins Hospital*, July, 1913.



18.—Aneurism of ascending and transverse portion of the arch of the aorta. (*Pennsylvania Hospital.*)



19.—Aortic aneurism a few days before rupture into the right bronchus. The ood can be seen trickling down over the lower part of the sac. (*Pennsylvania*

g. Very often that portion of the chest wall which overlies the n is destroyed completely so that nothing but the subcutaneous nd the skin remain. It is noteworthy that an aneurism does n rupture externally even when nothing but the skin intervenes.

has reported a case which showed an aneurism in its incipiency with the minute sac extending through the acutely broken and necrotic media to the adventitia. This, he believes, indicates that the aneurism may form in the acute stage of syphilis.

An aneurism is apt to develop under the following conditions: The aorta, most frequently the ascending portion of the arch, becomes the seat of a syphilitic aortitis. This lesion either weakens or destroys the media and inasmuch as the middle or elastic coat of the artery constitutes its real strength, a point of lowered resistance is established. If a sufficiently large area of the media is destroyed the vessel may dilate or bulge as the result of the strain of a normal blood-pressure. In the majority of the cases, as we have pointed out in the etiology, the occupation of the individual is such that the weakened wall is subjected to an extra strain because of a sudden elevation of the blood-pressure due to muscular effort. As a result of repeated strains of this sort the weakened point gradually stretches or the intima overlying the patch of mesaortitis may tear and hasten the formation of an aneurism.

If the entire circumference of the artery is diseased, the aorta usually undergoes dilatation, cylindrical or fusiform in shape. The extent of the dilatation varies greatly; it may be very slight or it may be so extreme as to constitute a true aneurism. Passive dilatation may occur as the result of a persistently high blood-pressure.

The most common type is that form in which the artery gives way at one side, constituting a bulging sac. As the aneurism increases in size the mouth of the sac becomes relatively small in comparison with the interior, the blood current in the sac becomes sluggish and a laminated clot is formed.

The rapidity with which the aneurism grows depends to some extent on the formation of a firm clot within the sac and the extent of the inflammatory reaction set up in the surrounding tissues, the latter tending to form a fibrous barrier about the tumor. As a rule, however, efforts toward arresting the increase in the size of the aneurism are futile unless the interior of the sac can be obliterated. As long as the mouth of the sac remains open, each succeeding systole tends to increase the strain on the weakened wall. The introduction of gold wire into the sac is done with the idea of aiding in the formation of a clot which will entirely fill it. In small arteries the dilated portion may become obliterated by the development of a new growth from the intima. In aneurism of the large vessels spontaneous healing rarely occurs, although instances of this have been noted, even in the thoracic aorta.

In the saccular form of aneurism there is usually no trace of either the intima or media, the wall being formed by the thickened adventitia or outer coat.

As the aneurismal tumor increases in size it gradually compresses the surrounding structures and may lead to their destruction. The aneurism may by pressure necrosis, erode through one or the other of the main bronchi, the superior vena cava, the trachea or the esophagus. The most striking result of the eroding effect of an aneurism, however, is to be seen in its destruction of tissues which resist its advance, such as bone. Thus it very commonly destroys part of the sternum and ribs as it extends forward or, if the descending aorta is involved, the vertebra may be destroyed. Figs. 418, 419, and 420 show the size a rapidly developing aneurism may attain. An aneurism differs from other tumors in that it is expansile and it is to this fact that its destructive ability is probably due. Not only does the tumor gradually increase in size but in addition the tissues with which it is brought into contact are subjected to an incessant

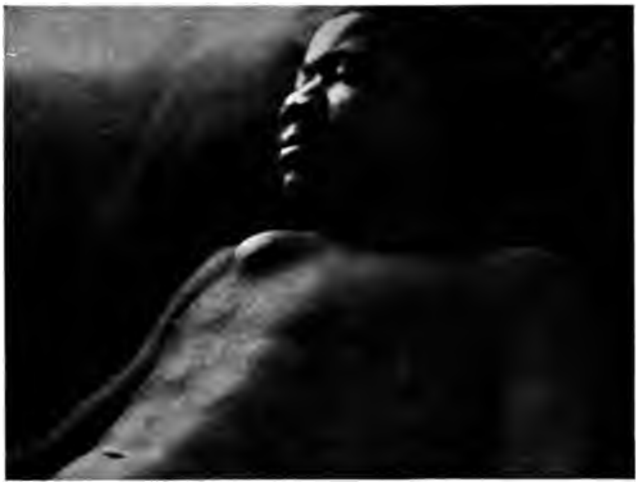


Fig. 418.—Aneurism of ascending and transverse portion of the arch of the aorta. (*Pennsylvania Hospital.*)

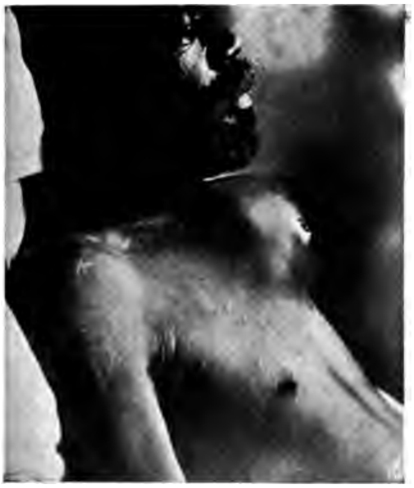


Fig. 419.—Aortic aneurism a few days before rupture into the right bronchus. The oozing blood can be seen trickling down over the lower part of the sac. (*Pennsylvania Hospital.*)

pounding. Very often that portion of the chest wall which overlies the aneurism is destroyed completely so that nothing but the subcutaneous tissues and the skin remain. It is noteworthy that an aneurism does not often rupture externally even when nothing but the skin intervenes.

While an individual with an aneurism may die from some intercu illness, in about one-half the termination is death from rupture o sac. Many cases of sudden death are due to rupture of an unsusp aneurism.

Usually there is but one aneurism but there may be two or mor Fig. 422). Occasionally there are a number of aneurisms invo different arteries throughout the body.

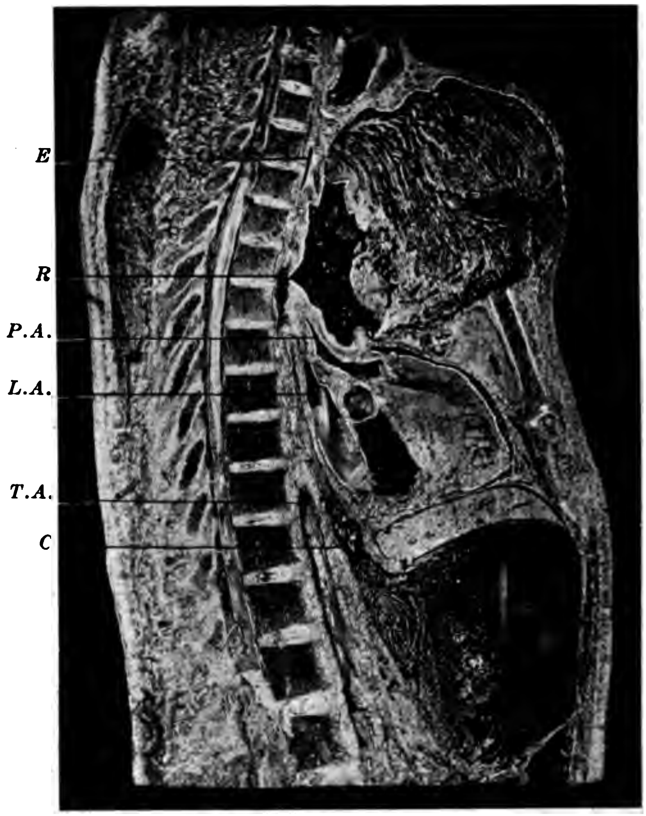


FIG. 420.—Sagittal section of aortic aneurism in case shown in Fig. 419. *E*, Es gus; *R*, point of rupture into right bronchus and esophagus. Immediately above this the bronchus is seen completely flattened out, accounting for the atalectatic left *L.A.*, left auricle; *T.A.*, thoracic aorta; *C*, cardiac end of stomach which is filled with clot; *P.A.*, pulmonary artery.

The aneurism has eroded the sternum and except for a small channel at its pos part is filled with a firmly organized thrombus.

The aneurism varies greatly in size. If of the dilatation type, lumen of the vessel may be slightly increased or the artery may be mendously distended in the form of a bag. In the more common sa lated variety the bulging point varies from the size of an English wa to an enormous tumor the size of a fetal head.

Symptoms of Aneurism in General.—The clinical picture of thor aneurism is subject to a number of variations depending largely on particular part of the arch affected. For this reason it seems best to describe the symptoms, pressure effects and physical signs of aneurism in general and then to group these findings around the various clinical types of aneurism of which they are characteristic.

Small aneurisms involving the intrapericardial portion of the aorta, and including the sinuses of Valsalva, are frequently of rapid growth and rupture may occur without there having been any previous symptoms (Fig. 423). Then, too, an aneurism may attain considerable size in the ascending aorta without causing symptoms, although physical signs may be well marked. The mobility of this part of the aorta and the fact that it is not in very close relationship with surrounding structures may account for the absence of subjective symptoms. On the other hand, aneurisms

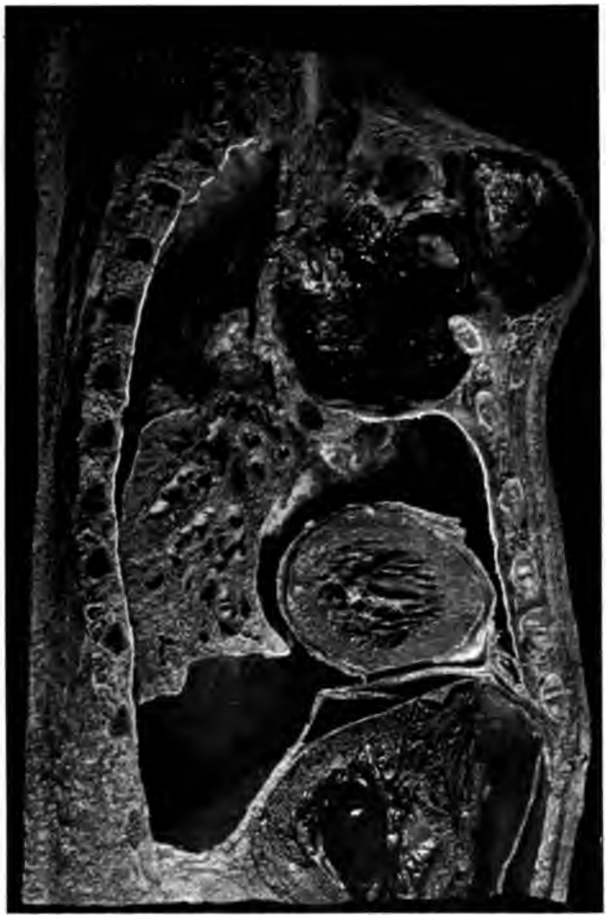


Fig. 421.—Aneurism of the aorta, hydrothorax, atelectasis of the left lung. The atelectasis resulted from pressure on the bronchus; the hydrothorax from pressure on the pulmonary veins as shown in Fig. 420. The frozen serum has been removed from the pleural cavity, and the laminated clot, from the aneurismal sac.

that give rise to numerous and severe symptoms may not be demonstrable by physical signs; this is quite apt to be the case when the aneurism involves the transverse or descending portions of the arch. This is due to the fact that the transverse arch is so firmly fixed and in such close relations with important structures as the trachea, esophagus, left bronchus, left recurrent laryngeal nerve, etc., that even a small aneurism in this situation is apt to cause marked pressure symptoms.

The descending portion of the arch is also apt to manifest itself by symptoms rather than physical signs. Recognizing these facts, Broadbent divided thoracic aneurisms into two classes, namely, ***aneurism of physical signs*** and *aneurism of symptoms;* the former term applying to aneurism of the ascending and the latter to aneurism of the transverse and descending portions of the arch.

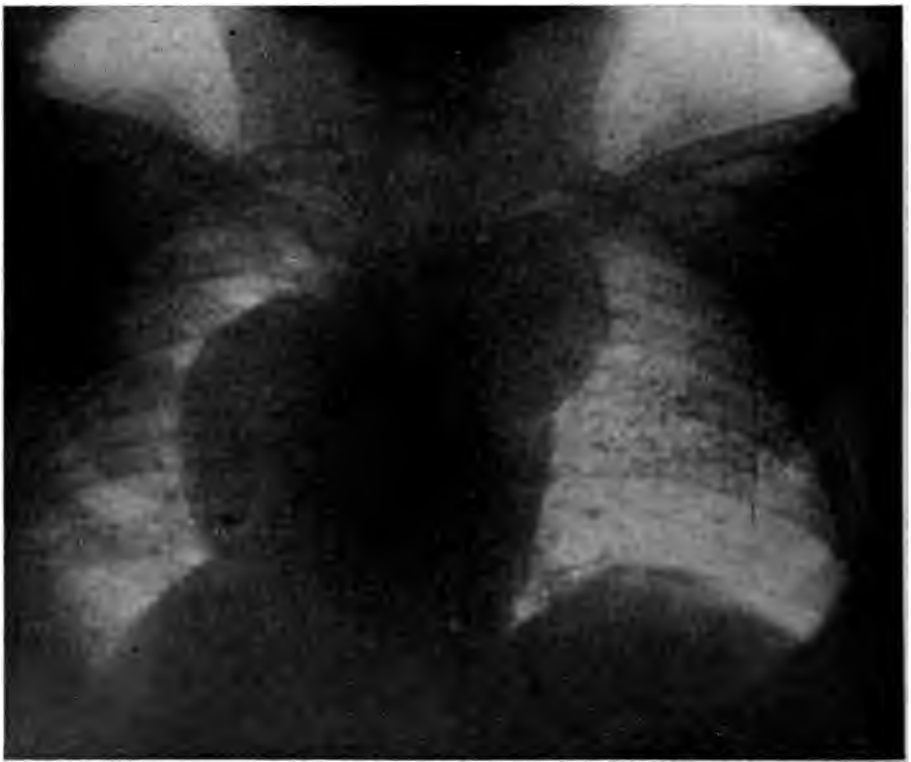


FIG. 422.—Large multiple aortic aneurism. (*Courtesy of Dr. H. K. Pancoast.*)

As a rule patients suffering from aneurism are, when first seen, robust, vigorous-looking young and middle-aged individuals. In the beginning the symptoms become noticeable only upon exertion when there may be some dyspnea, palpitation of the heart, a sense of precordial oppression or actual pain.

Pain is one of the most distinctive features of aneurism. It may occur as the result of disease in the aorta itself or it may be due to the pressure of the growing tumor. The latter will be considered under pressure effects.

Pain referable to disease of the aorta may be due to one of two causes: (1) It may be anginoid in character due to involvement of the coronary orifices as the result of an aortitis and this pain may recur in the form of attacks of true angina pectoris. (2) Osler has called attention to the fact that many individuals at the onset suffer from attacks of pain of the greatest severity, this being due to splitting of the intima over a patch of mesaortitis. Attacks of this character gradually disappear as the tumor increases in size.

In about one-half of the cases perforation of the aneurism causes death before there has been any marked deterioration in the general health. When death is not caused suddenly by perforation, the patient gradually fails in strength, the disease pursuing a course similar to that seen in chronic heart disease. Aortic insufficiency is very frequently a marked feature of aneurism and as a result the left ventricle hypertrophies; later, when compensation fails, there develop marked dyspnea, edema and other evidences of cardiac failure. In some cases a severe secondary anemia develops as the result of oozing from the aneurism. An unusual event is perforation of the aneurism into the vena cava; death usually occurs within a few hours but the patient may live for some months.

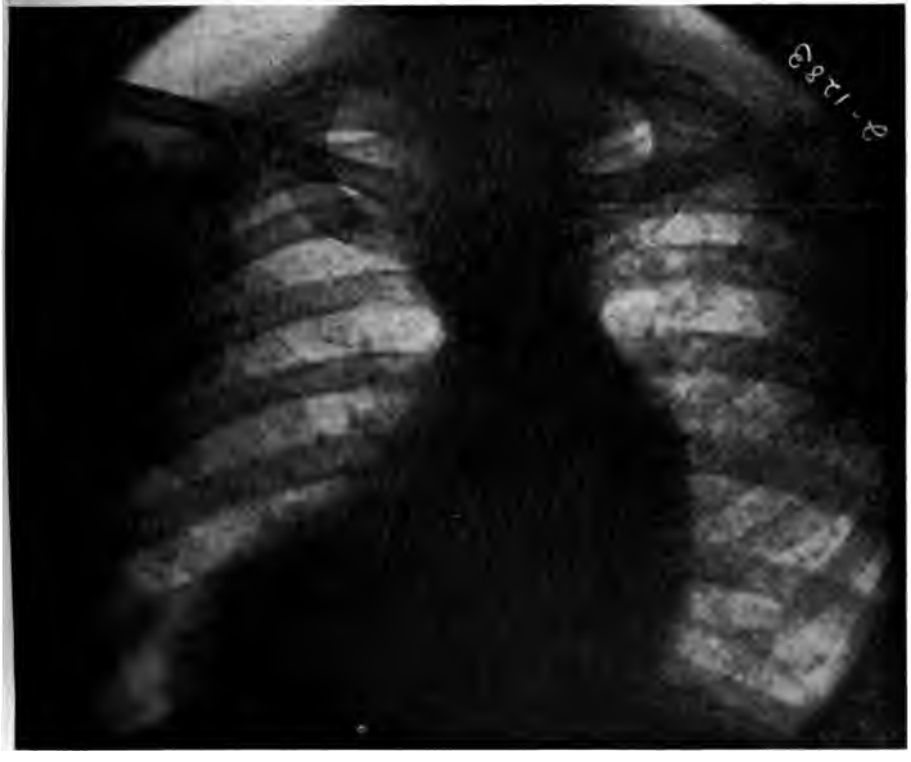


FIG. 423.—Aortic aneurism (intrapericardial). (*Courtesy of Dr. H. K. Pancoast.*)

The terminal event may be an attack of pneumonia or death may occur as the result of compression of the esophagus, trachea or a main bronchus

Dieulafoy has emphasized the importance of *tuberculosis* as a terminal infection in cases of thoracic aneurism, having noted it in 18 of 46 cases under his observation.

Pressure Effects.—*Pain.*—In the majority of aneurisms involving the aorta pain is at some time or other a conspicuous symptom. The character of the pain varies greatly in different cases. It may be nothing more than a transient feeling of substernal discomfort or it may be in the nature of persistent or recurring attacks of neuralgia. Keeping in mind this fact, one should always be alert to the possibility of a thoracic aneurism in individuals who complain of chest pain which is not otherwise explainable.

Pain produced by an aneurism in the ascending aorta is, as a rule, not severe in spite of the fact that it erodes the sternum and ribs. Pain is more apt to occur as the result of an aneurism in this situation when t[illegible] growth extends backwards. It is usually vague and not at all seve[illegible] except when the aneurism is growing. The pain may be increased [illegible] relieved by different postures; or it may be referred to the right shoulder [illegible] the scapular region. Spasm of the right trapezius muscle has been note[illegible] also.

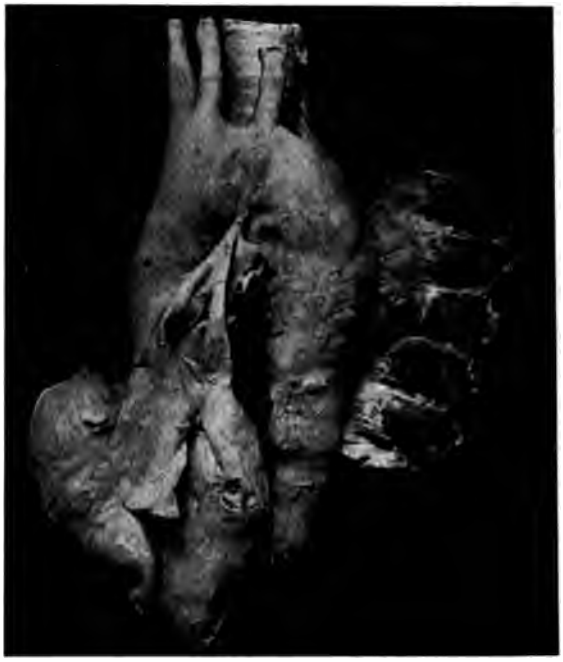


FIG. 424.—Aneurism of ascending transverse and descending aorta, the latter be[illegible] adherent to the dorsal vertebræ which were in part eroded from pressure.

When the transverse portion is involved, the pain is usually vague an[illegible] deep-seated. In this situation the growth is more apt to interfere wit[illegible] the function of the structures immediately surrounding it rather than gi[illegible] rise to severe pain.

The most intense pain is produced by an aneurism involving the descending portion of the arch and the descending thoracic aorta. This is due to the close relation of the spinal column and the thoracic aorta (Fig. 424). In this situation erosion of the vertebræ, the implication of the intercostal nerves and, in some cases, the spinal cord often cause a persistent, boring pain or sharp, lancinating neuralgic attacks of the greatest severity. The pain may be the only manifestation of the disease.

Special Nerves.—Next to pain the most common manifestation of pressure is the involvement of certain special nerves.

Left Recurrent Laryngeal.—This nerve, which arises from the vagus, enters the thorax, passes around the arch of the aorta and ascends in the space between the esophagus and trachea. In addition to supplying motor fibers to the larynx it also sends branches to the esophagus, trachea and pharynx. It can be readily seen that if the aneurism involves the transverse portion of the arch there is very apt to be a disturbance of function in the parts enervated by the left recurrent laryngeal. If the nerve is stimulated, there may be produced painful spasms of the pharynx, the esophagus or larynx. In addition there may be difficulty in swallowing (dysphagia) or a feeling of suffocation due to narrowing of the glottis. The nerve may be so pressed or stretched as to cause paralysis of the left vocal cord; in such instances the voice may be husky, cracked or tremulous in character. The so-called "*brassy*" *cough* is frequently present (see Figs. 92 and 170).

The right recurrent laryngeal nerve does not enter the thorax but curves around the subclavian artery. It is sometimes involved by extension of the aneurism upward to the right or as the result of the innominate also being involved.

Rarely the *phrenic nerve* is implicated. When this occurs there may be hiccough or even paralysis of the diaphragm.

Sympathetic Nerves.—Inequality of the pupils in cases of thoracic aneurism may be due to several causes. Pressure on the sympathetic nerves is usually credited with being the most common cause of the condition. The pupil on the affected side may dilate or contract according to whether the sympathetic fibers are irritated or destroyed by the aneurism. In some instances the inequality may be due to changes in the blood-vessels, compression or obstruction in the carotid causing a dilated pupil. It is to be borne in mind that inasmuch as thoracic aneurism is so frequently a luetic manifestation, the pupillary change may be specific in origin and independent of the aneurism; when a manifestation of syphilis, the light reflex is lost, whereas in cases of aneurism the sensitiveness to light is retained.

Esophagus.—Difficulty in swallowing may be transient or persistent. When transient, it is usually due to spasm resulting from irritation of the recurrent laryngeal nerve; when persistent, it is due to direct pressure of the aneurism (see Fig. 420). This may occur when either the transverse or descending portions of the arch are involved; it is occasionally noted in aneurisms of the descending thoracic aorta. The pressure is rarely sufficiently marked, however, to cause complete blocking of the esophagus but there may be sufficient narrowing of the lumen to render the swallowing of solid food difficult or impossible. In considering the possibility of stricture of the esophagus the presence of an aneurism must be excluded before attempting to pass a stiff exploring instrument, otherwise the growth may be perforated.

Trachea and Bronchi.—When the aneurism occurs in the transverse portion of the arch, pressure on the trachea is apt to occur. This may give rise to a dry, brassy cough; there may be in addition some expectoration which is often blood-streaked. Dyspnea may also occur. If the trachea is directly compressed, there may be little difficulty in breathing except when the patient exerts himself. In some instances the dyspnea may be paroxysmal in character and be so intense as to cause a feeling of suffocation. These paroxysmal attacks may be due to direct compression of the trachea or as a result of the left recurrent laryngeal nerve being involved. More rarely one of the main bronchi may become compressed (see Figs. 420 and 421). When either the trachea or bronchi are involved there is apt to be some diminution in the intensity of the respiratory murmur; this will be bilateral if [illegible] th[illegible] trachea is compressed and unilateral if the lumen of one of the m[illegible]a[illegible] bronchi is narrowed.

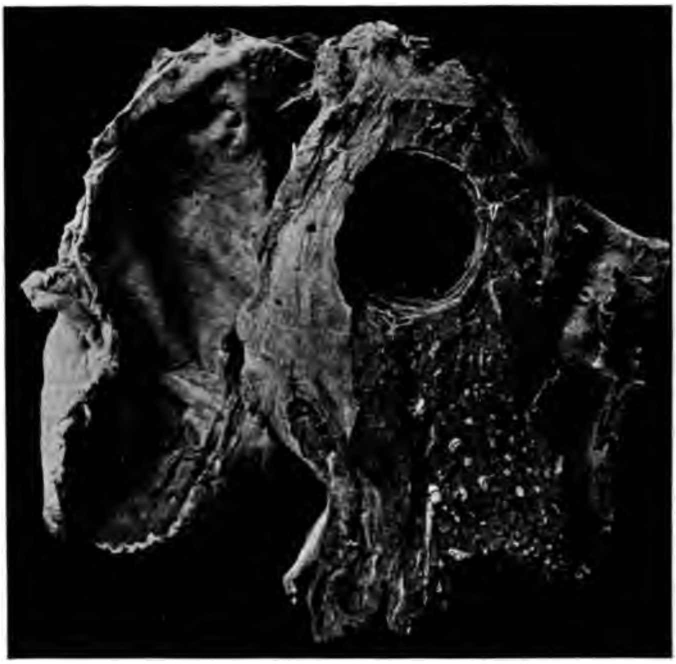


FIG. 425.—Left lung is greatly compressed in its upper part. Circular opening sh[illegible]ws interior of the aneurism sac. Note large size of aneurism and small size of heart. (*Je[illegible]fer-son Medical College Museum.*)

The Lungs.—In some instances one or the other of the lungs beco[illegible] compressed by the aneurismal sac. The lung immediately adjacent [illegible] the aneurism becomes congested and atelectatic and may eventua[illegible]l undergo fibroid changes. Bramwell[1] has reported 13 cases of ao[illegible]t aneurism in which pressure at the root of the lung produced consoli[illegible]da tion or chronic pneumonia. In 8 cases the left lung was involved, i[illegible] 4 the right. Tuberculosis was present in 1 case, side not given. A weakened respiratory murmur and hemoptysis are apt to result if the compression is at all marked (Fig. 425).

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[1] *Edinburgh Med. Jour.*, xvi, No. 2, 1916.

Veins.—Among the most striking effects of aneurism is compression of one of the great veins. The veins most likely to be involved are the inferior vena cava, the innominates, the subclavians and the pulmonary veins. Complete obliteration of the superior cava is rare but a certain amount of compression is not unusual. If the compression occurs suddenly, it gives rise to varying degrees of edema of the head, neck and arms and cyanosis of the face. Rarely the larynx and pharynx may become edematous and thus give rise to severe dyspnea.

When the pressure is exerted gradually, the collateral veins assume much of the burden and thus obviate the symptoms described above. But even when an adequate collateral circulation is established, there is very apt to be some congestion of the veins of the head and neck and sometimes one or other of the arms. The jugulars are often distended. Distention of the jugulars under these circumstances is to be distinguished from that occurring in cases of failing compensation by reason of the fact that the veins do not pulsate in cases of aneurism.

The collateral circulation may be carried on through the intercostal and internal mammary veins; through the deep cervical, the vertebræ and intercostal veins; and through branches of the mammary veins on the surface of the chest. The front of the chest may be occupied by a mass of dilated veins, some of which are the size of a finger. In some instances the veins are in the subcutaneous tissue; in others the veins of the skin itself are greatly distended. As a rule, dilated veins are not seen over the lower part of chest but occasionally they are, and in some cases they may extend over the abdomen.

When one or the other of the innominate veins is involved independently of the superior vena cava, the obstruction manifests itself by a one-sided distention of the veins of the neck. In the same way distention of the veins of one upper extremity may occur if the subclavian vein on that side has been compressed.

An unusual occurrence is pressure on the right pulmonary veins. This gives rise to congestion of the lung and later to a hydrothorax (Fig. 109). The mechanism is the same as when the distended right auricle compresses the right pulmonary veins and thus leads to an effusion.

Symptoms of Rupture.—The terminal event in about one-half of the cases of aneurism is a fatal hemorrhage due to rupture of the sac. Lemann[1] collected 592 cases of thoracic aneurism in which rupture occurred. The rupture involved the following structures respectively in the order of their frequency: Pericardium alone 148, left bronchus, pleura or lung 160, right bronchus, pleura or lung 62, esophagus 50, trachea 48, superior vena cava 31, pulmonary artery 18, into other structures 40. Although rupture externally through the skin is looked upon as being quite rare, Lemann found 35 cases (5.9 per cent.) recorded in which the accident had occurred.

The occurrence of hemoptysis in association with aneurism is not infrequent. The sputum may be blood-streaked as the result of an erosion in the trachea, one of the main bronchi or the lung itself. As in the case of rupture into other organs an immediately fatal hemorrhage

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[1] *Am. Jour. Med. Sc.*, August, 1916.

can be recognized at a glance, but in the absence of this
almost certainly occur if the examination is not thoroug
inspection of the patient is an absolute necessity and if thi
not carried out carefully and systematically the clue leadin
diagnosis will often escape notice. "How many eyes—ye
eyes, too—looked at the thorax and never saw the so-called
or diaphragm phenomenon? Many of us look at it every d
see it, even after we know about it. How often does the
thoracic aneurism go begging for want of a careful glanc
McCrae). There are two prime essentials: (1) The pati
stripped to the waist; and (2) there must be a good light.

Inequality of the pupils is always suggestive of some
mischief and should therefore be given due consideration.
ance in the return of the blood through the superior cava
tributaries may be suggested by congestion of the face or
nence of the veins in the neck or upper extremities. The dis
veins may be bilateral or unilateral. In some cases the v
anterior chest wall may be distended also; sometimes to a
degreee.

In addition to distention of the veins one or both of the
swollen and congested or pallor and sweating may be presen
and arm as the result of pressure on the sympathetic. In a
unilateral swelling, simulating clubbing of the fingers, has

When the chest itself is subjected to inspection, the pa
placed facing a good light. The observer should inspect t
different positions; in some cases a slight pulsation become
when an oblique light falls on the chest (see Fig. 116).

One of the most important, if not the most important, di
of thoracic aneurism is the presence of a pulsation in the a
base of the heart. Fig. 426 indicates the points at which
to aneurism is most frequently noted. In addition, when t
thoracic aorta is involved, pulsation may be noted in the left

to one side or the other of the median line. Osler gives the following description of impulses which might be mistaken for aneurism: (1) The throbbing of the conus arteriosus in the second left interspace—very common in young persons and in thin chests, and seen particularly well during expiration. (2) Pulsation of the heart in the second, third, and fourth left interspaces, extending as far as the nipple in cases of fibrosis and retraction of the upper lobe of the lung. (3) Cardiac pulsation in the second, third and fourth right interspaces in association with fibrosis and retraction of the right upper lobe. (4) Effusion on either side of the chest may so displace the heart that there is a marked impulse at or outside the nipple line on either side. (5) Throbbing subclavians seen in the outer half of the infraclavicular regions; this is seen in thin-chested persons, in neurasthenia, Graves' disease, early tuberculosis and anemia. In some instances, when the pulsation is unilateral, it may be accompanied with a thrill and a murmur forming a mimic or phantom aneurism. (6) In the back of the chest a visible pulsation is nearly always aneurismal; but occasionally, in Broadbent's sign, the tugging may be so limited and localized in one interspace that it simulates pulsation, but palpation easily corrects this.

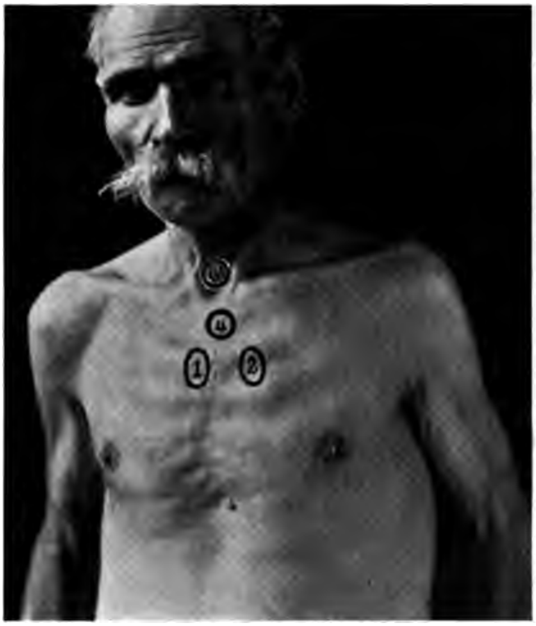

Fig. 426.—Locations on the anterior chest wall at which aneurismal impulses may be noted in their order of frequency.

In some cases of anemia, in Grave's disease and neurasthenia there may be a diffuse throbbing over the interior portion of the thorax or a distinct systolic impulse may be noted in the lower part of either side of the chest. A pulsation due to a purulent pleural effusion is occasionally seen. It is usually on the left side and occurs below and outside the nipple.

Except the pulsation that occurs in the back, the farther removed the impulse is from the areas shown in Fig. 426 the less likely is it to be due to aneurism of the aorta. In association with the impulse the ribs to the right or the left of the sternum or the manubrium itself may project abnormally as the result of the underlying sac or there may be a distinct tumor if the bony structures have become eroded. The skin over the tumor may be ulcerated or may "sweat" blood (Fig. 427).

The apex beat of the heart is often displaced downward and outward, partially as the result of pressure and partially because of cardiac hypertrophy. The heart may, however, show no enlargement and occupy its normal position (see Fig. 425). The heart is said not to be enlarged unless the aortic valves are incompetent.

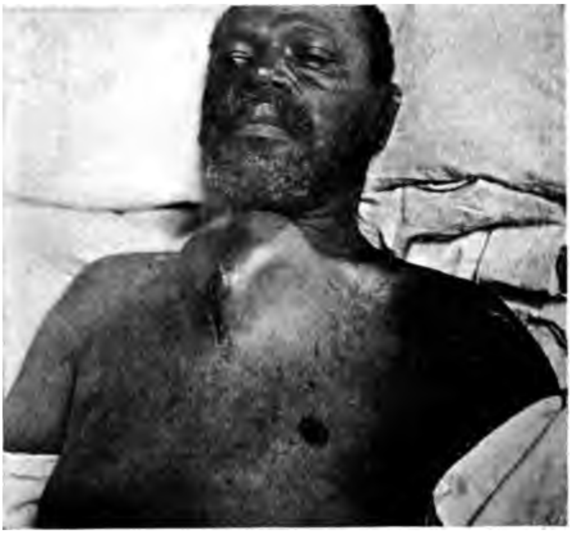

FIG. 427.—Oozing of blood from aneurism sac.

In every case of suspected aneurism the larynx should be inspected. Pressure or stretching of the left laryngeal nerve may lead to paralysis of the abductor muscles and consequently to paralysis of the left vocal cord. As a rule this leads to some dyspnea and alterations in the voice but even with the vocal cord fixed in the middle line, there may be no change in the voice.

Rarely the right laryngeal nerve may be involved and still more rarely there may be bilateral abductor paralysis.

Palpation.—Three important signs are obtained by palpation:

1. *Diastolic Shock.*—This is one of the most conclusive signs of aneurism, some observers ranking it as the most important single sign we have. It consists of a short, sharp impulse coincident with diastole.

2. *Thrill.*—This is usually systolic in time; rarely it is double or diastolic alone. If the sac contains much clot, and especially if the anterior portion of the sac is filled, the thrill is usually absent. It occurs more frequently in cases of dilatation of the arch than in the sacculated variety of aneurism.

3. *Pulsation.*—If the aneurism has perforated the chest wall there will be imparted to the palpating hand an impulse which is heaving and expansile in character. If the pulsation is noted to be expansile there is little doubt as to the true nature of the trouble. This may be absent, however, if the sac is filled with a clot. In some cases the sac is soft and fluctuating but usually it is firm in consistency. In the early stages of aneurisms which eventually perforate and in those which are deep-seated and never appear externally, the impulse may be very slight. Bimanual palpation should always be practiced in suspected cases. One hand is placed over the base of the heart and the other over the vertebræ and firm pressure is made. This will often reveal a slight pulsation which would otherwise escape notice. As a rule the impulse is best felt by placing the palm of the hand flat over the suspected area but in some cases the impulse can be detected only by pressing the finger tips deep into the intercostal spaces. The impulse may be felt only at the end of expiration. The throbbing of an aneurism differs from that felt over a hypertrophied heart in that it is localized and imparts to the palpating hand a heaving and expansile quality. In some cases the impulse is felt only when the fingers are pushed downward in the suprasternal notch.

Tracheal tugging or Oliver's sign may be elicited in cases of aneurism of the transverse arch when the sac has become adherent to the trachea. This is apt to produce a downward tug of the larynx with each impulse. The sign may be elicited by having the patient sitting or standing in front of the observer who gently but firmly grasps the cricoid cartilage with the thumb and finger while the head is slightly extended. The head is then flexed and the patient told to hold his breath. If a tug is present, the up-and-down motion of the larynx is transmitted to the observer's fingers. Another way of detecting this sign is for the examiner to stand behind the patient, steadying the latter's head against his body. The cricoid cartilage is then firmly grasped by the thumb and forefinger. Instead of an up-and-down motion of the larynx there may be a lateral movement. This is known as Cardarelli's sign.

The Peripheral Arteries.—In most individuals suffering from aneurism the arteries are palpably thickened, but in young syphilitic subjects the arterial degeneration may be limited to the aorta.

The interposition of an aneurismal sac between the heart and the peripheral vessels may lead to retardation or feebleness of the pulse in the latter. The effects of the sac on the pulse wave are: (1) to delay it; (2) to diminish its height; (3) to cause its duration to be longer, and the subsidence more gradual and slower than normal; and (4) the artery will also be constantly full between beats, as the sac acts like a blacksmith's bellows—being, in fact, a reservoir of blood (Broadbent).

If the sac is located in the ascending portion of the arch, the alteration in the pulse wave will be the same on both sides and there is then no standard of comparison, but if the sac involves the great vessels given off the transverse portion of the arch, the radial and carotid pulses on one side may differ from those on the opposite side.

If, for instance, the innominate artery is compressed or its opening is partially blocked by clot, the right radial pulse will be smaller than the left. In the same way the opening of the left subclavian may be involved, thus giving rise to a smaller pulse in the left radial.

If, on the other hand, the sac is interposed between the innominate artery and the left subclavian and carotid arteries, there will be a delay in the appearance of the left pulse. The carotid pulse will often show the same changes. It is thus seen that inequality or retardation of the pulse is a valuable sign. Simultaneous tracings of the two radials will often show the most marked differences in cases of aneurism. Inequality of the radial pulses alone can occur in conditions other than aneurism, such, for instance, as an anomalous radial artery on one side, the pressure of a tumor on the subclavian or axillary artery, or blocking of the orifice of the innominate or left subclavian artery in cases of extensive atheromatous disease of the aorta.

Percussion.—Unless the aneurism is very small or deep-seated, dulness on percussion can be elicited over that portion of the chest wall which overlies the arch. The areas over which dulness is most commonly encountered correspond to those in which pulsation is noted, namely, the second and third interspaces just to the right and to the left of the sternum and over the manubrium. Dulness over the dorsal vertebræ may be present, even early in the disease.

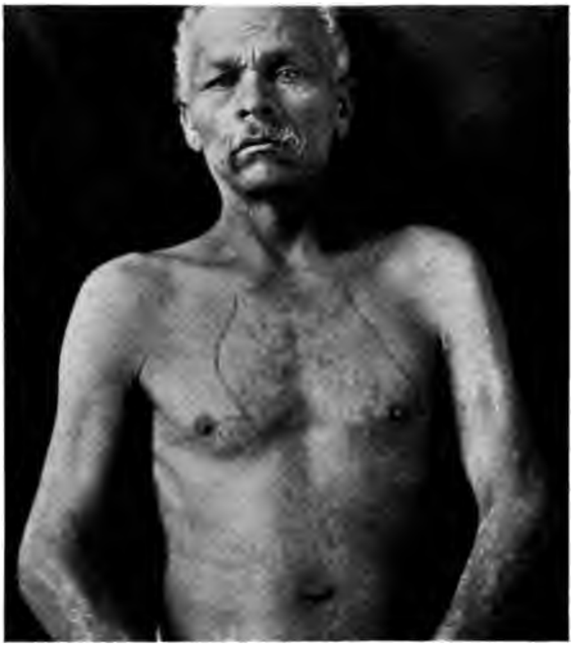

FIG. 428.—Aortic aneurism (syphilitic) in mulatto, aged 62. Shortness of breath on exertion; chest pain. Physical signs of aortic and mitral insufficiency.

There may be a small area of marked dulness at one of these points and beyond an area of relative dulness. In cases of dilatation of the aorta, dulness may extend across the sternum and for some distance on either side, at the level of the second and third interspaces (Fig. 428). If a tumor is present, the size of the sac may be determined, approximately, by the area of relative dulness which extends around the tumor. In cases of aneurism of the descending thoracic aorta, dulness may be detected in the left interscapular region or about the angle of the left scapula. The occurrence of localized pain and dulness over the painful area is suggestive of aneurism even if other symptoms and signs are absent. Occasionally percussion over the upper part of the sternum is accompained by pain or a severe paroxysm of coughing when there is no other evidence of aneurism (Eichhorst).

Percussion of the cardiac area will often show no enlargement of the heart, although it may be displaced downwards and to the left by the aneurismal sac. Hypertrophy is most apt to occur if the patient is also the victim of a general arterio-sclerosis, Bright's disease or aortic regurgitation.

Auscultation.—The auscultatory signs in aneurism are not distinctive and are relatively of less importance than the signs elicited by the other procedures. Very often the heart sounds are as distinctly heard over the aneurism as over the body of the heart itself, giving the impression of there being a second heart. A murmur, usually systolic in time, is often heard; the murmur may be diastolic in time, especially if the first portion of the arch is involved or there may be a to-and-fro murmur. Probably the most distinctive auscultatory sign is the presence of a ringing and sharply accentuated second aortic sound. The farther this accentuated second sound is heard from the aortic cartilage, the more significant it becomes. Even with the presence of a diastolic murmur the second sound is rarely entirely replaced. If the unaided ear or a rigid stethoscope is used, the diastolic shock may be felt coincidently with the hearing of the accentuated second sound.

The heart itself may show no abnormality. In the terminal stages, however, there may be the evidences of failing compensation.

Pressure on the trachea may cause a weak and feeble respiratory murmur over both lungs. If the pressure is exerted on one of the main bronchi, usually the left, the respiratory murmur on the corresponding side may be less intense than over the opposite lung. Complete occlusion of a main bronchus will cause an entire absence of breath sounds over the affected lung.

Blood-pressure.—If there is present a widespread arterio-sclerosis or Bright's disease marked arterial hypertension may occur but in the majority of cases of thoracic aneurism the blood-pressure is normal. If, however, the pressure is taken in both arms there may be quite a marked difference in the two sides; this has the same significance as inequality or retardation of the pulse wave, as determined by the palpating finger. Pressure differences of 5 mm. are common in arteriosclerotic subjects without aneurism. In the latter condition unilateral differences of 10 to 20 mm. are not rare.

X-ray Examination.—So thoroughly have the X-rays demonstrated their usefulness in mediastinal affections that no examination in case of a suspected aneurism can be considered as being complete without their employment. Even in cases in which the physical signs and symptoms leave little room for doubt, the X-rays enable us to determine the extent and the character of the aneurism and whether there is more than one portion of the aorta which is involved. The existence of not a few aneurisms is first revealed as the result of a fluoroscopic or radioscopic examination which has been made for some other purpose.

Classification of Aneurisms Involving the Thoracic Aorta.- *Aneurism of the Intrapericardial Portion of the Arch and Sinuse Valsalva.*—When the thoracic aorta is involved, the aneurism most quently occurs in the ascending portion of the arch. This is due to fact that a syphilitic mesaortitis is so commonly limited to the first port of the vessel. It may involve one or all of the sinuses of Valsalva. this situation the aneurism is usually small, and deeply placed; i frequently first discovered at the autopsy table or as the result of an X- examination (see Fig. 423). During life the clinical picture is apt to that of angina pectoris or aortic insufficiency. This is due to the f that the exciting cause of the aortitis may lead to a partial occlusion of orifices of the coronary arteries or it may produce aortic incompeten either by dilating the aortic ring or by extension of the disease to the va leaflets. In the presence of either one of these conditions, therefore, aneurism should be suspected. An aneurism in this situation may co press the superior vena cava and thus cause congestion of the face undue fulness of the jugulars and the veins of one or other upp extremity.

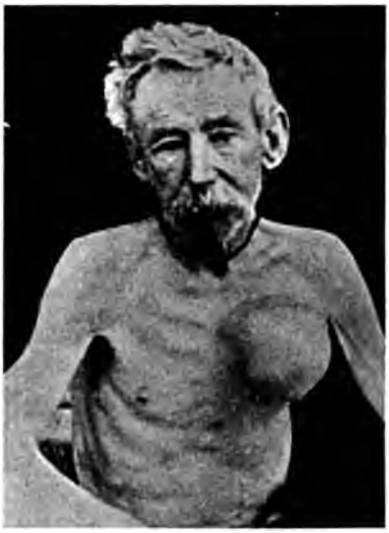

FIG. 429.—Large aneurismal tumor presenting to the left of the sternum.

2. *Aneurism of Ascending Arch.*—This includes that portion of t aorta between the sinuses of Valsalva and the orifice of the innomina artery. An aneurism in this situation commonly attains a very large si and this may occur without symptoms, or the symptoms may be ve trivial in nature. For this reason an aneurism affecting this portion of t aorta has been called the aneurism of physical signs. The heart is oft dislocated downward. The aneurismal tumor extends upward and the right appearing in the second and third interspaces to the right of t sternum (see Fig. 418); it may, however, appear to the left of the sternu (Fig. 429). Varying degrees of pain may occur, especially when the ri and sternum are being eroded. Dyspnea, substernal oppression or p

are apt to be increased by exertion. Pressure symptoms are relatively infrequent in this situation but compression of the superior cava may occur as shown by distention of the veins and congestion of the head, neck and upper extremities. Rarely the inferior vena cava may be compressed, giving rise to edema of the feet and legs.

3. *Aneurism of the Transverse Portion of the Arch.*—This is the aneurism of symptoms. The transverse portion of the aorta is not only firmly fixed but is in close relations with several important structures. Behind it lie the trachea, esophagus and thoracic duct; below is the left bronchus; and curving around it is the left recurrent laryngeal nerve. Being deeply placed an aneurism may develop in the transverse arch without giving any physical signs. On the other hand, pressure symptoms are usually marked as the close relation of various important structures renders them liable to compression even by a relatively small aneurism. The aneurismal tumor may extend in any direction but usually it grows backward; it may, however, project forward presenting a little to the right or left of the median line.

In aneurisms of the ascending portion of the arch the pulses are equal as the sac is located on the proximal side of the innominate and left carotid and subclavian arteries. When the transverse arch is the seat of the aneurism, either the orifices of the main bronchus may be included in the sac or the bulging may take place between the innominate artery and the left carotid and subclavian arteries. In either case an inequality of the pulse waves is produced in the two radial and carotid arteries. An aneurism in this situation does not, as a rule, attain a very great size and a fatal termination is apt to occur at a much earlier period than in the case of aneurisms of the ascending portions of the arch.

Pulsation may be absent but often the presence of a slight impulse can be detected by making firm pressure over the manubrium with the palm of the hand. Dulness may be noted in the same situation. An aortic systolic murmur and an accentuated second sound are frequently heard.

Pressure symptoms are common and may be the only evidence of an aneurism. Thus there may be pain or difficulty in swallowing; dyspnea, which may be paroxysmal in character; hoarseness; a brassy cough; and hemoptysis.

Not infrequently the ascending and transverse portions of the arch are involved together.

4. *Descending Portion of the Arch.*—This portion of the aorta lies to the left side of the dorsal vertebra from the third to the sixth. The only important structures near it are the esophagus and left bronchus. An aneurism in this situation usually grows backward and erodes the vertebra or it may compress the esophagus and rupture into it. There is a paucity of both symptoms and physical signs in an aneurism in this location. If one has reason to suspect a thoracic aneurism the left interscapular region and the area about the angle of the left scapula should be carefully examined as a tumor sometimes appears in these locations.

Commonly pain is the only manifestation. This may be of a dull aching character or a sharp, shooting neuralgic pain. The pain may be localized at one point along the vertebral column or it may radiate around the body and down the left arm and is often intense. The condition is often mistaken for Potts' disease. Musser states that an aneurism in this situation often simulates phthisis very closely. The fact that tuberculosis is not uncommon in those subject to aneurism of the aorta often masks the presence of the latter.

5. *Descending Thoracic Aorta.*—This portion of the aorta extends from the sixth to the twelfth dorsal vertebra where it becomes continuous with the abdominal aorta. An aneurism in this portion of the aorta is not common and is frequently latent. It usually occurs near the diaphragmatic opening. The aneurismal tumor usually extends backward and often erodes the vertebra producing severe pain. In some cases the sac becomes enormous and may present externally in the left back (see Fig. 430). It may, however, project forward and if located just above the diaphragmatic opening cause epigastric pulsation.

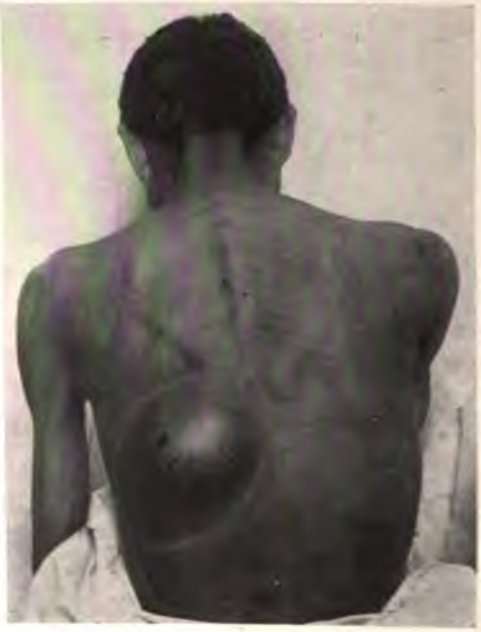

FIG. 430.—Aneurism of the descending thoracic aorta.

Dysphagia sometimes occurs but, as a rule, the only symptom is pain. This varies greatly. It may be very slight or it may be persistent and of the most excruciating character.

Diagnosis.—Not the least remarkable thing about thoracic aneurisms is the frequency with which they can occur without giving the faintest symptom or sign to indicate their presence. In view of the size of some of them it is almost inconceivable how they can exist in this silent manner. There is nothing I know of which is so conducive to the bringing about of a chastened spirit as the demonstration of a large aneurismal sac by the roentgenogram or autopsy in a case in which the idea that such a condition was present had never occurred. There is this much to be said in extenuation, however, an aneurism of considerable size may exist in which neither physical signs nor pressure symptoms can be elicited even after its presence has been clearly shown by the fluoroscope and radiogram. Two such cases have been seen recently in the University Hospital.

Again in cases of a diastolic or double aortic murmur, with a positive Wassermann reaction, the presence of an aneurism involving the intrapericardial portion of the aorta, is always to be thought of. An aneurism in this situation rarely gives any clue as to its presence other than occurrence of these murmurs.

In the majority of cases the first evidence of trouble is a sense of substernal oppression or actual pain especially after exertion. If there are associated with the pain pressure symptoms or physical signs, the existence of an aneurism is always to be thought of. The symptoms and the location and nature of the physical signs have been fully dealt with and need not be discussed here.

The three conditions which are most apt to be confused with aneurism are: (1) a mediastinal tumor; (2) pulmonary tuberculosis; and (3) mitral stenosis.

While a *mediastinal tumor* bordering the vertical axis of the chest may give rise to signs they are more apt to occur in or extend to areas not involved by a thoracic aneurism. The tumor, as a rule, does not perforate the chest wall nor is it associated with expansile pulsation, a ringing second aortic sound or diastolic shock. The presence of arterio-sclerosis and a positive Wassermann reaction are points in favor of the trouble being an aneurism.

At first sight there seems no good reason why *phthisis* and aneurism should be confused. When we recall, however, that dyspnea, hoarseness, chest pain, cough, hemoptysis and loss of weight are common in both and that dulness may extend upward toward the apex of the lung or unilateral suppression of the breath sounds may occur in aneurism, one can understand how mistakes occur. In aneurism there is either no fever or it is very slight. In addition physical signs of vascular disease are usually present in aneurism and the sputum does not contain tubercle bacilli. It must not be forgotten, however, that the two conditions are not uncommonly associated.

Although paralysis of the left vocal cord as the result of pressure on the recurrent laryngeal nerve in cases of *mitral stenosis* is not common, it must be kept in mind as the paralysis, hoarseness and a brassy cough have led to a diagnosis of aneurism. Careful auscultation of the heart will usually prevent such a mistake.

SPECIAL FORMS OF ANEURISM

DILATATION OF THE AORTA

By many, simple passive dilatation of the aorta is not considered as being a true aneurism. If, however, we embrace under the term "aneurism" cases in which the lumen of the arterial tube is uniformly expanded, simple dilatation must be included. The condition really represents an incipient form of aneurism and while it lacks some of the features of the saccular variety, it has many symptoms and physical signs in common with that type of the disease.

Etiology.—There are two forms of dilatation of the aorta, namely, *passive* and *dynamic.* The former is due to a gradual increase in the diameter of a portion of the aorta or even the entire arch, the lumen of the to the fact that, as a race, they are extremely prone to devel tive changes in the arteries as the result of a syphilitic i McCrea's series of 35 cases no less than 14 were Negroes.

From X-ray studies Smith and Kilgore[1] determined that of the aorta might be increased, in extreme instances, to ne normal size as the result of: (1) syphilitic aortitis; (2) arteri individuals over fifty years of age; (3) chronic endocardit in the relatively young; and (4) arterial hypertension.

Among the more infrequent etiological factors may be m acute infectious diseases, especially acute rheumatic fever, Graves' disease.

Morbid Anatomy.—The primary cause in nearly all cases aorta, the dilatation being a secondary manifestation. Th dilate at any point but the most frequent site is the ascendi the arch which is also most subject to atheromatous and syph (Fig. 431).

Passive stretching of the aorta may be caused, occasio result of a blood-pressure which persistently remains hig instances the intima may be smooth or show but little ev endarteritis deformans.

The dilatation may be cylindrical or fusiform in shap instances the great vessels given off from the arch are also

When the first portion of the aorta becomes dilated, th may also be involved thus giving rise to aortic insufficiency hypertrophy. This is especially apt to occur when the ao syphilitic in origin. Other organic heart lesions, either muscular, are common.

Symptoms.—Dilatation of the aorta may be present symptoms, or the symptoms may be so trivial as not to attra In the latent form the condition is usually an accidental dis post-mortem room. Now that the X-rays are being used for diagnostic purposes many examples of a dilated aorta a light which otherwise would have been unsuspected. Furt more exact knowledge which now prevails as to the etiolo valvular lesions is directing more attention to the aorta.

In the cardiac form there may be attacks of faintness; shortness of breath on exertion, cough, and, finally, all the symptoms of broken compensation. The most frequent cardiac lesion is aortic insufficiency, the leakage being due in some instances to disease of the aortic leaflets, in others to stretching of the aortic ring.

Physical Signs.—*Inspection.*—In old people with rigid chest walls there may be no visible impulse. When present, the impulse may be most noticeable in the suprasternal notch; it also may be noted in the second and third interspaces to the right of the sternum or over the manubrium. When the aorta becomes dilated the main branches are sometimes lifted upward so that pulsation in the right subclavian is seen above instead of below the clavicle.

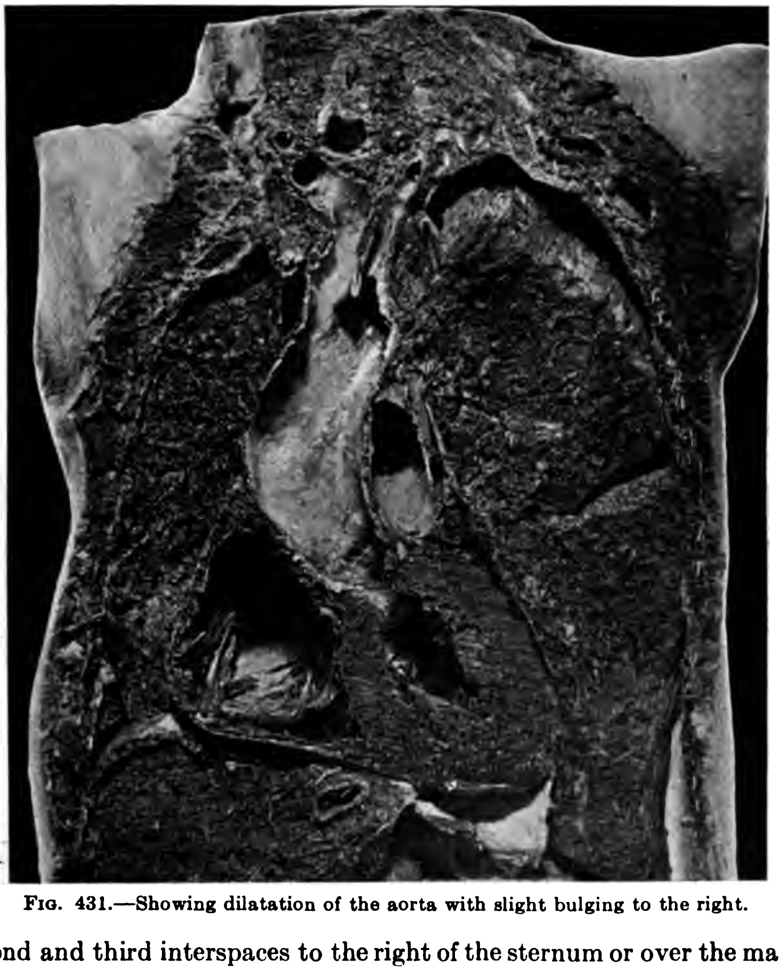

FIG. 431.—Showing dilatation of the aorta with slight bulging to the right.

Palpation.—In the absence of visible pulsation the impulse may be felt by pushing a finger downwards in the suprasternal notch or by applying the palm of the hand firmly over the manubrium. A systolic thrill localized over the aortic area and a diastolic shock in the same region are not infrequent. If aortic insufficiency is present, there may be a diastolic thrill. Except in those cases in which dilatation has been produced by arterial hypertension the blood-pressure is, as a rule, low.

Percussion.—Dulness over the manubrium above the level of the second ribs may be caused by extension of the aorta upward; this is the most constant single sign (Osler). Lateral expansion of the artery may be indicated by dulness in the second and third interspaces to the right or left of the sternum. Owing to the frequent association of aortic insufficiency with dilatation of the aorta the area of cardiac dulness is often greatly increased (Fig. 432).

Auscultation.—A systolic murmur having its punctum maximum at the aortic cartilage and transmitted to the carotids, is often heard. This murmur is generated in the dilated portion of the aorta as the result of the blood being thrown into swirls. Projecting calcareous plates may also aid in producing the murmur. The second sound is sharply accentuated and ringing or clanging in character. This serves to distinguish the systolic murmur from that occurring in aortic stenosis in which the second sound is very faint or not heard at all. If aortic insufficiency is present, there is also a diastolic murmur which may occur alone or in association with the systolic brûit. In aortic insufficiency due to stretching of the aortic ring, the diastolic murmur rarely replaces entirely the ringing second sound as is apt to be the case when the leakage has been caused by acute endocarditis. Another point of difference is that the diastolic murmur occurring with dilatation of the aorta is often transmitted upward.

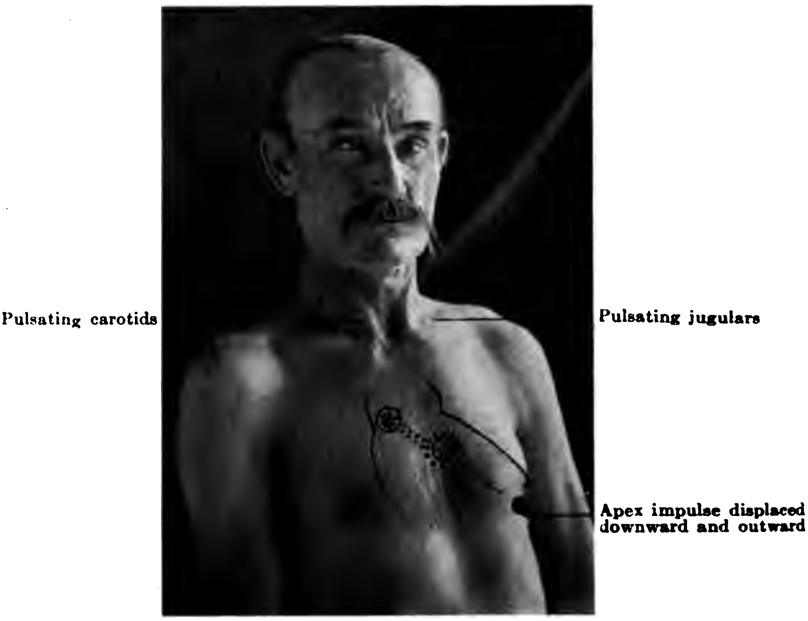

FIG. 432.—Physical signs in aortic insufficiency due to dilatation of the aorta. The dots indicate the diastolic murmur.

ARTERIO-VENOUS ANEURISM

Communication between an artery and vein is not an infrequent occurrence in the peripheral vessels and is usually the result of a trauma. Gunshot wounds, especially those produced by the modern type of bullet, are the most common causes of this accident.

Involvement of the large vessels in the thorax is relatively rare. The aneurism may perforate the superior vena cava, one of the pulmonary arteries or one of the auricles.

Superior Vena Cava.—An aneurism of the ascending portion of the arch sometimes ruptures into the vena cava. As a rule, death occurs in a few hours but the patient may survive for months.

When the perforation occurs, the patient is suddenly seized with intense dyspnea, the face becomes cyanosed and the veins in the neck greatly distended. In the course of a few hours a tense edema of the face and neck appears. Later the veins over the chest become enlarged from collateral circulation established between the internal mammary, the intercostals and the epigastric veins. The enlargement of the veins terminates abruptly at the level of the diaphragm. Over the site of the perforation, in the area about the aortic cartilage, there may be a marked thrill. In addition there is a continuous, roaring murmur with systolic intensification.

According to Osler the only other condition which gives rise to sudden cyanosis of the head and neck is a severe, crushing accident to the thorax.

Pulmonary Artery.—Perforation into the pulmonary artery is attended with sudden and severe dyspnea followed by cough and occasionally with blood-streaked sputum. A thrill over the pulmonic area is occasionally present. The most characteristic physical sign is a continuous, roaring murmur, with a systolic intensification heard over the pulmonic area. Perforation of the pulmonary artery is not, as a rule, immediately fatal. A period of weeks or even months may elapse before death occurs.

Auricles of the Heart.—Death usually occurs within a few minutes. There may be dyspnea, cough and cyanosis, but in most of the reported cases there have been no characteristic symptoms.

ANEURISM OF THE INNOMINATE ARTERY

When the transverse portion of the arch is implicated, the first part of the innominate is frequently included in the aneurismal sac. When such is the case, the only clinical manifestation of involvement of the innominate is a diminution or retardation of the pulse wave on the right side. The innominate may be involved either independently of, or in association with an aneurism of the aorta.

The principal symptoms occurring in an aneurism of the innominate artery are dyspnea, pain and a throbbing sensation at the root of the neck. Dysphagia may also occur.

As a rule, the aneurismal sac presents behind or just above and to the right of the right sterno-clavicular joint. The sac usually extends forward and not infrequently causes a dislocation of the right sterno-clavicular joint. The right radial pulse is retarded or less full than the

the arch by reason of the fact that the latter rarely extend the clavicle. If an aneurism occurs in both situations, the innominate may be overlooked unless the tumor is plainly v

In both dynamic and passive dilatation of the arch of t subclavian is not infrequently lifted up so that there is a m tion just above and to the right of the sterno-clavicular joi sometimes referred to as a phantom aneurism. Furtherm tively frequent finding is the presence of a systolic murmur clavian arteries. It is practically always functional in char thought to have some connection with an apical tubercul facts must be kept in mind when considering the possibility In those instances in which the subclavian artery is lifted result of dilatation of the aorta there is no difference in the pulses.

RUPTURE OF THE AORTA

Etiology.—Rupture of one of the coats of the aorta may healthy vessel as the result of a fall or a severe blow on the occurrence of such an accident is rare. Although cases have b in which rupture of a healthy aorta is said to have follo muscular effort, the weight of authority is against the probab an accident. It is more than likely that in all such cases the some degeneration of the aorta.

In the vast majority of cases of rupture of the aorta the v come the seat of atheromatous or syphilitic disease. When any unusual muscular strain may cause the intima to split. stances the intima may be torn while the patient is at rest: i an abnormally high blood-pressure may be the exciting caus

Morbid Anatomy.—I have already emphasized the fact t mary lesion in degenerative diseases of the blood-vessels o media and that for some time the intima may present a nor ance. The lesion in the media, however, constitutes a point resistance and any abnormal strain within the vessel may ca lying intima to split. In the most severe forms of rupture all

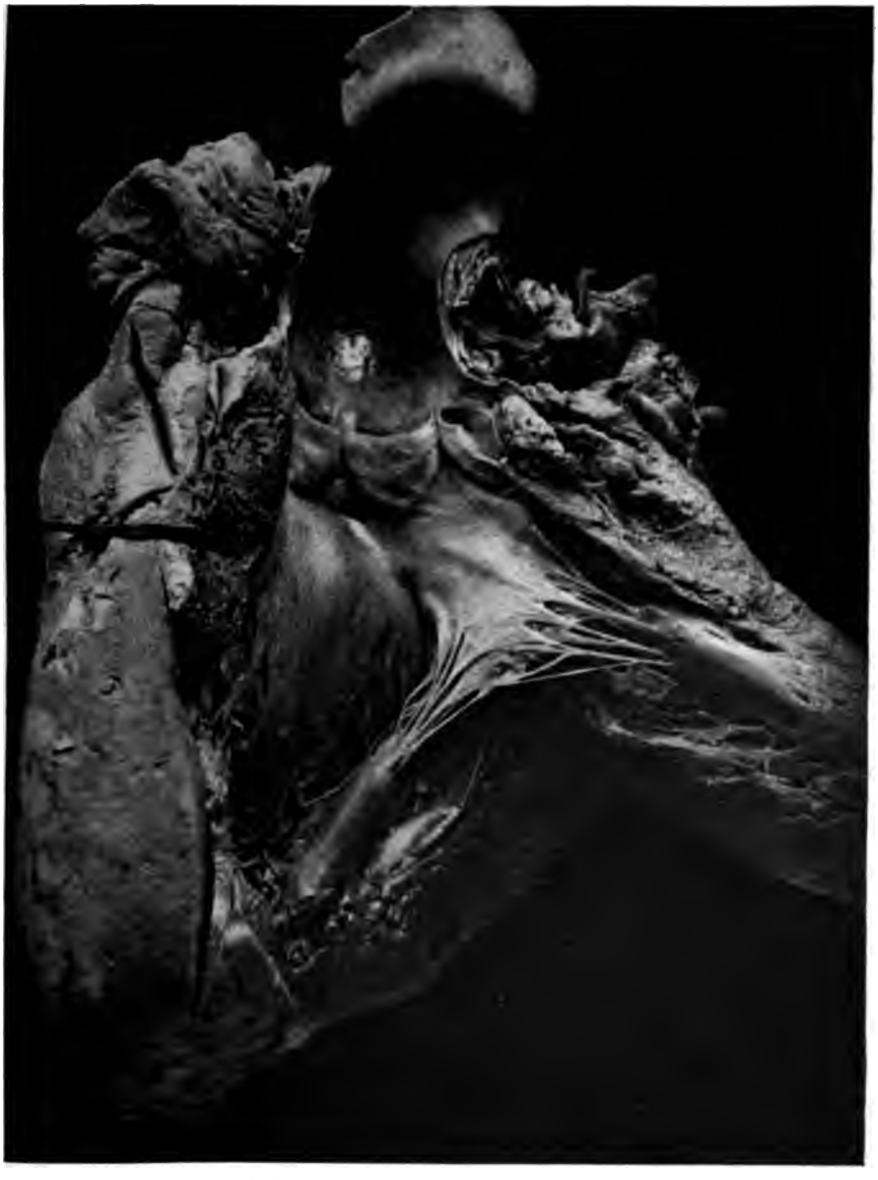

Fig. 433.—Rupture of the aorta. **Clinical Notes**: Sudden death without premonitory symptoms during convalescence, six days after an operation for hemorrhoids. **Pathologic Notes**: Two and one-half centimeters above the attachment of the aortic valves there is a *rough, slightly shaggy looking tear running directly across the aorta.* It is 2 cm. in length and leads directly from a small aneurismal pouch into the pericardial cavity. The surface of the aorta along the ascending arch shows small, scattered, yellowish nodules. *(Specimen from the Pennsylvania Hospital. Pathologist: Dr. G. C. Robinson.)*

media and adventitia and form what is known as a dissecting aneurism. In such cases death may occur at once; or the blood may break through the media and intima at a lower point and again communicate with the lumen of the vessel; or external rupture may occur at some future time. A few cases have been recorded in which the dissecting aneurism healed.

The most frequent site of the rupture is in the first or ascending portion of the aorta (Fig. 433). There are two reasons for this: (1) the greater frequency of syphilitic and atheromatous disease in this situation; and (2) the blood, as it is forcibly expelled from the ventricle at each systole, rapidly expands the aorta. If, therefore, the vessel wall is weakened by disease, a rupture of the intima may readily occur and especially after unusual effort. The tear in the intima may be transverse or vertical and is usually clean cut.

Symptoms.—In many cases death is instantaneous and the true nature of the trouble is first revealed at the autopsy table.

In those instances in which death does not occur immediately, pain is the most prominent symptom. This may be substernal and of moderate severity. It is not infrequent in the early stages of aneurism, probably as the result of tearing the intima. Later this pain disappears.

In those cases in which external rupture takes place the initial tear in the intima and media is attended by severe substernal pain and the signs of collapse. Death may occur at once. Not infrequently, however, there is an interval which may last from a few hours to two weeks when the patient is again seized with pain and almost immediately expire

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